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**A SHORT
PRACTICE OF MEDICINE**

A
SHORT PRACTICE
OF
MEDICINE

BY
ROBERT A. FLEMING

M.A., M.D., F.R.C.P.E., F.R.S.E.

LECTURER ON PRACTICE OF MEDICINE, SCHOOL OF THE ROYAL COLLEGE,
EDINBURGH: ASSISTANT PHYSICIAN, ROYAL INFIRMARY, EDINBURGH.

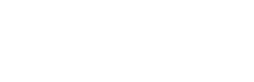


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PREFACE

THE writing of a new book on the Practice of Medicine demands an apology, or at least an explanation. There are many excellent text-books, but there are few small-sized manuals which fill the place in the library which the present volume is intended to occupy.

Medical science has made such strides in the direction of specialism and along the lines of every system, that the time must come when students attending a class on general medicine will be armed with a text-book giving the general outline of each individual disease, and thus only special points, clinical cases in illustration, and hints on treatment will be left to be written down. Such a plan saves much pen-driving, and frees the teacher from dictating year in and year out statements which can be as well, if not better, mastered beforehand from a text-book.

Further, the student, burdened with innumerable classes, demands some guide as to what is necessary and what unnecessary for the all-important examinations which lie before him, and such a guide is offered in the following pages. For the busy practitioner it may also not be out of place to provide a volume which, if it does not exhaust the historical account and theories of every disease, contains a résumé of all that is most important.

No attempt has been made to group together clinical features in tabulated form, because such tables are more useful if drawn up by the reader himself, but a definite method has been followed, where possible, so as to aid the student's memory in mastering the clinical features of each disease.

It has been found impossible in the space at disposal to write, as originally intended, a brief account of the medical anatomy and physiology of each system, but diagrams have

been used to elucidate the difficulties of the structure and functions of the nervous system.

The methods of qualitative and quantitative testing of the normal and abnormal constituents of urine and stomach contents have been omitted because they appertain more properly to the manual of clinical medicine, and for similar reasons pulse tracings with their diagnostic significance have not been included.

While the idea with which the book has been written is that everything should be mastered by the reader, the results of paralyzes of spinal nerves have been given in detail for purposes of reference.

I am indebted to many writers from whose works hints and help have been obtained, and the experience gained during past years in the Edinburgh Royal Infirmary has not unprofitably aided the descriptions of certain diseases.

I have also to thank for friendly counsel and advice several of my colleagues on the staff of the Edinburgh Royal Infirmary with whom I have discussed numerous difficulties. In particular I should mention Mr. J. J. Graham Brown and Mr. C. Wilson whose ready criticism of several special sections has been most helpful, and Mr. F. C. Nicholson to whom I am indebted for reviewing the proofsheets.

The plates and cuttings have been derived from various sources, and I wish specially to thank Professor Osler who has permitted me to quote several diagrams from his well-known text-book on the *Brain and Spinal Cord*. Lastly, I desire to thank the services of Mr. J. Grive, who is responsible for the reproduction of the illustrations.

J. FLEMING.

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SPECIFIC INFECTIOUS DISEASES

I. THE FEVER PROCESS

FEVER is pyrexia, and implies chemical changes occurring in the body, by means of which the organism as a whole suffers more or less markedly. The results of the fever process may be classified as follows:—

Table I.

Page 376, line 21, for "Fibroid Degeneration of the Uterus"

"Fibroid Degeneration of the Uterus"

Page 436, line 20, for "Chronic Tuberculosis of the Lung"

"Chronic Tuberculosis of the Lung"

I. Classification based on aetiology.

Exogenous fevers are as a rule dependent on the action of a specific micro-organism, and to this type of fever the term "idiopathic" is sometimes been applied.

2. *Pyæmic fever* is the result of the introduction into the blood-stream of definite pyæmic organisms.

3. *Septic fever* is due to the introduction of the chemical products of special organisms, as well as the organisms themselves, into the system.

4. *Hectic fever* is due to the diffusion into the blood-stream the morbid products of organisms.

5. *Symptomatic fever* is the term applied to pyrexia ending on a local injury or local inflammation: urethral catheter fever is a good example of this type.

II. Classification based on Types of Pyrexia.—A recent classification depends on the type of pyrexia present, the following types may be mentioned:—

1. *Continued fever* is pyrexia, in which the temperature remains for a certain number of days at a heightened level.

2. *Intermittent fever* is pyrexia, in which the fall of temperature, generally occurring during the day, reaches the normal.

3. *Remittent fever* is pyrexia, in which several rises of temperature occur, but the intervening fall does not reach normal.

4. *Relapsing fever* is a type of pyrexia in which periods of heightened temperature are succeeded by periods of apyrexia.

5. *Hectic fever* implies generally a nightly rise succeeded by a morning fall of the temperature to or about normal, and is usually associated with great sweating.

The normal skin temperature is stated as 98·4° or 98·6 F., but it is invariably higher in the mouth and rectum by half a degree to a degree. In old persons the temperature is slightly subnormal. The daily variations must be remembered. There is a trivial rise in the afternoon between 3 and 6 P.M., and a fall in the morning between 3 and 5 A.M.

There are heat-centres, probably situated in the medulla, which regulate in a remarkable way the temperature of the body. Heat is lost by the skin and lungs, and by an elaborate reflex mechanism the supply of blood to the skin may be made greater or less by these heat-centres. There is not merely loss of heat from a dry skin, but sweating, with the resultant evaporation of water, causes very rapid loss of heat. To keep, therefore, a fair equilibrium, there are constrictor and dilator centres which govern the amount of blood sent to the skin and the secretion and excretion of sweat; and these centres are affected by the increased distribution of blood for special purposes to the stomach and other abdominal organs.

The mode of heat production has given rise to considerable differences of opinion, and naturally our explanation of the fever process is closely associated with the accepted theory. In the fever process is it the case that, according to Traube, (1) heat is generated to the same amount but not discharged; or (2) is there increased production, the discharge remaining the same; or (3) is there increased production far in excess of the increased discharge? Experimental evidence suggests that heat production is under the control of the nervous system, and that the thermogenetic centres are situated, as already stated, in the medulla.

Clinical Features.—There is increased tissue waste, with defective excretion by the skin, kidneys, and bowels, and therefore a tendency to the retention and accumulation of waste products in the system.

Alimentary System.—The tongue is furred and dry, sordes are present in the mouth, there is marked loss of appetite, failure of the digestive processes, excessive thirst, and not infrequently vomiting. Constipation is the rule, although in certain fevers diarrhœa occurs. The liver is frequently swollen and painful.

Hæmopoietic System.—The red blood corpuscles suffer both numerically and as regards their hæmoglobin, while in some cases there is a leucocytosis and the spleen is acutely congested. In some fevers the lymphatic glands are inflamed.

Circulatory System.—The heart's action is excited, the pulse full and bounding but also dicrotic, although in the late stages of many long-continued fevers degenerative changes occur in the myocardium, and thrombosis may occur in the veins.

Respiratory System.—The breathing is rapid, but agrees in ratio with the frequency of the heart's action, unless, as in certain cases, hypostatic congestion and other pulmonary complications or sequelæ supervene.

Integumentary System.—The skin is usually hot and dry, but in some types of fever, such as rheumatic fever, sweating is a special feature. Many fevers are characterised by definite rashes. In the late stages of any debilitating fever bedsores are apt to occur.

Urinary System.—There is a diminished amount of urine, which deposits urates on standing, and is of high colour and specific gravity. There is often a deficiency of chlorides; and albumin, with tube-casts, may be present. Bladder difficulties, especially in connection with loss of consciousness, are common in severe cases of fever. Suppression of urine occasionally occurs.

Reproductive System.—Menorrhagia is frequent, and abortion is induced where the pyrexia is high, should pregnancy fortunately complicate the case.

Nervous System.—Malaise is typical, and it includes headache and general discomfort, which in great measure is refer-

PRACTICE OF MEDICINE

Sleeplessness, convulsions, delirium, coma, and other features of severe types of fevers; while the term *hyperpyrexia* is meant a temperature exceeding 104° and is included under nervous phenomena.

Muscular System.—The muscles rapidly waste, and there is no growth in the more serious fevers.

Stages of Fever.—Most of the eruptive fevers pass through stages, which include:—

1. *Stage of Incubation*, during which there are no

2. *Stage of Invasion*, during which the first clinical signs, including malaise, and generally more or less

3. *Stage of Advence or Eruption* is characterised by the appearance of the rash, where a rash is present, and the symptoms referable to the fever process are at their

4. *Stage of Defervescence or Resolution*, during which the fever either suddenly by *crisis* or gradually by

5. *Stage of Convalescence*, during which, in a favourable case, recovery occurs.

Complications or *sequela* may delay a favourable issue. In malignant types of fever death may occur at almost any stage, even when incubation is over. It sometimes happens that a severe febrile fever kills the patient before any eruption

Treatment of the Fever Process.—It is prudent

in the treatment of a continued fever by careful regulation of the alimentary canal, and a purge is therefore

indicated. Relief may also be obtained by stimulating the bowels to act freely. A hot bath and a dose of

nitrosi render the patient much more com-

fortable in fever is most important. As a general rule

the chief part of a typical fever dietary, and if

it is taken, beef-tea and soups of a simple nature

are in place. In not a few cases alcohol is a

valuable aid, but it should rarely be given until other measures

have been tried. It will support life, but its administra-

tion should be deferred, if possible, until the more acute stage

of the fever is passed. In alcoholic subjects its use is obviously objectionable.

The room selected for the fever patient should be light and airy, and where it is an infectious disease, all unnecessary furniture, hangings, and carpets should be removed. Insist on having a hair mattress, or, where necessary, a water-bed, but on no account permit a feather-bed to be used. In the more severe cases of a continued fever it is a great comfort to the patient to have two beds of similar height in the sick-room, so that he can be moved from one to the other occasionally. The bed should be placed in such a position that free access can be obtained to it. The utmost care should be taken to have sheets and blankets absolutely clean; and where the excreta are infective, antiseptic precautions must be taken so as to prevent any danger to others, and especially to the nurses and attendants. Carbolic acid, chloride of lime, and many other powerful antiseptics can be used for sterilising stools, etc. A good nurse is even more important than a carefully appointed sick-room; and, where necessary, night and day nurses should be procured. A most important point is to insist on the nurse carefully noting the amount of food given and the hours at which it has been administered; and, similarly, medicines and stimulants should be charted in an orderly and exact fashion.

It is hardly requisite to describe here the treatment of all the different symptoms of an ordinary fever, but it is desirable to refer in detail to the method of treating hyperpyrexia. Every fever patient should, if possible, have the skin sponged at least twice a day, as the temperature, even when tepid water is used, is considerably reduced by this procedure.

The following methods, in order of increasing severity, may be employed for the treatment of hyperpyrexia:—

1. Sponging the patient with tepid water or eau-de-logne and water.

2. Sponging the patient with cold water or iced water, taking care to dry each part of the body that has been sponged before proceeding to the next part.

3. Wrapping the patient in a sheet or blanket wrung out cold or even iced water. For this procedure the use of two sheets is almost imperative. A waterproof sheet is spread on the bed, a dry blanket is placed on it, then the sheet or

blanket wrung out of cold or iced water, in which the patient is wrapped, and blankets placed on the top. At intervals the temperature should be taken in the mouth so as to avoid too great reduction, and on no consideration should the temperature be brought down to normal. Before removing the patient to a dry bed care must be taken to prevent collapse by giving a stimulant when necessary.

4. The patient may be placed in a bath, the temperature of which should be about 80° Fahrenheit, and then by adding cold water or iced water it can be rapidly reduced to 50° or 60° Fahrenheit. A more severe method of treatment is to pour cold or iced water over the patient's head and back, whilst in the bath. Generally fifteen to thirty minutes is the maximum of the patient's endurance, and every precaution should be taken to prevent collapse, as already mentioned.

Many patients owe their lives to treatment on one or other of these lines, and the cold-bath or hydropathic treatment of typhoid fever has proved most successful, although from a patient's point of view it may be a great hardship.

No drugs, which can be wisely given, do much for the reduction of temperature, and on no consideration should antipyrene, antifebrin, or other antipyretic remedies of this group be administered for this object, because of their depressing effect on the heart, which the hydropathic treatment most certainly obviates. Perhaps the only drug which is an antipyretic, and which it is prudent to administer, is quinine in doses of 10-15 grains at bedtime, although its use is limited to certain fevers only.

In any long-continued fever bedsores are apt to occur. They may be guarded against by attention to cleanliness and by frequently washing the skin with soap and water. Hardening the skin with methylated spirit or whisky rubbed into, and allowed to dry on, the part is very useful, and a dusting powder of zinc oxide or boracic acid and starch may be freely employed.

II. TYPHOID FEVER

(ENTERIC FEVER; TYPHUS ABDOMINALIS)

AN infectious fever characterised by a special involvement of Peyer's patches and solitary glands in the intestine, with

usually the appearance of a limited rose-coloured rash, enlargement of the spleen and mesenteric glands, and, in most cases, typical temperature chart.

Etiology.—The typhoid bacillus of Eberth measures 3 to μ in length by 1 μ in breadth, is flagellated and actively motile, and is found in the stools, the urine (25 to 30 per cent of cases), the blood, the spleen, and, it is said, in the joints. Less commonly it occurs in endocarditic vegetations, pleural and other exudates, and in meningeal effusions. It stains with aniline (basic) dyes, and in one of the rounded ends there is a refractile body which was thought, at one time, to be a spore.

As a result of the presence of the typhoid bacillus, the body manufactures an antitoxin which interferes with the motility of the bacilli. Widal's reaction consists in the action of the blood of a patient suffering from typhoid upon a culture of the typhoid bacillus in bouillon of twenty-four hours' growth, and it should produce rapid clumping of the organisms with cessation of movement. A dilution of 1 part of the patient's blood to 30 of sterile bouillon may be taken as the minimum standard, and the period of time within which this change should occur should not exceed sixty minutes. A typhoid patient gives a typical reaction after one week of the disease.

The disease is commoner before middle life. It is especially frequent in hot, dry weather, and is the direct result of the typhoid bacilli infecting the water-supply, milk, food, particularly oysters, vegetables which are eaten uncooked, such as lettuce, and ice, in which the bacilli for a long time retain their vitality.

We are still uncertain as to the limit of time during which the bacilli remain active. The following figures are interesting: in garden earth 21 days, in filter-sand 82 days, in street dust 30 days, and on clothing 60 to 70 days. During the South African War many typhoid cases were unquestionably the result of inoculation of food by flies. It is extremely improbable that the organisms are transmissible in air.

The stools of a typhoid patient almost certainly increase in volume during the process of decomposition.

Pathological Anatomy.—The intestinal lesions are the most typical.

During the *first week* Peyer's patches and the solitary glands of the lower part of the small intestine, and probably at first close to the ileo-cæcal valve, undergo enlargement and become of a pink or fawn-coloured appearance. There is not merely hyperæmia, but there is a rapid increase of lymphoid cells.

At the beginning of the *second week*, if resolution does not commence (an abortive attack), necrotic changes occur.

During the *third week* ulceration of these necrosed patches commences, although it is most probable that even during the preceding week this process may have begun.

The ulcers are bile-stained, do not necessarily involve the whole of a Peyer's patch, and the depth varies in different cases. Sometimes the floor of the ulcer is the muscular coat, in more severe cases the peritoneum, with naturally an increased risk of perforation. The edges of the ulcer are usually swollen and often undermined. The base of the ulcer is smooth and clean. As already indicated, the older ulcers are to be found in the lower parts of the ileum.

Towards the end of the third week cicatrization occurs, and, depending upon the amount of destruction, there may be more or less constriction of the bowel. The mesenteric glands are always inflamed, of reddish colour, and in certain cases they may suppurate and so cause peritonitis. The large intestine is not so frequently or so markedly affected, but the cæcum naturally suffers to a greater extent than the colon. The jejunum may be involved right up to the duodenum, but the process is much less extensive than in the ileum.

The possible results of ulceration may be perforation, severe hæmorrhage, and in rare cases stricture from cicatrization.

The spleen is invariably enlarged and acutely congested, occasionally infarction occurs, and in rare cases the spleen may rupture. The liver and kidneys show cloudy swelling, and hypostatic congestion of the lungs occurs before death in fatal cases. In certain cases the bacillus attacks the endocardium or pericardium, the pleura, the cerebral meninges, and other parts, considerably modifying the clinical features from the usual type.

Clinical Features.—The stage of *Incubation* is one to two weeks. The premonitory symptoms consist in lassitude, headache, nausea, epistaxis, bronchitis, sometimes abdominal pain, or even a rigor.

During the *first week* the temperature steadily rises until it reaches 103° or 104° F. Each evening the record is higher than the one preceding. The pulse is rapid, full, and of low tension. There is generally constipation, the tongue being white and coated, and about the end of the first week the rash may appear. The rash, consisting of rose-coloured spots, comes out first on the abdomen, and usually the spots number from six to twenty at a time. They are slightly raised, but disappear on pressure. They are, as a rule, confined to the trunk, and in about one-fifth to one-quarter of all cases they are absent altogether. The spleen becomes enlarged about the same time, and there is usually nocturnal delirium.

At the beginning of the *second week*, during which necrosis

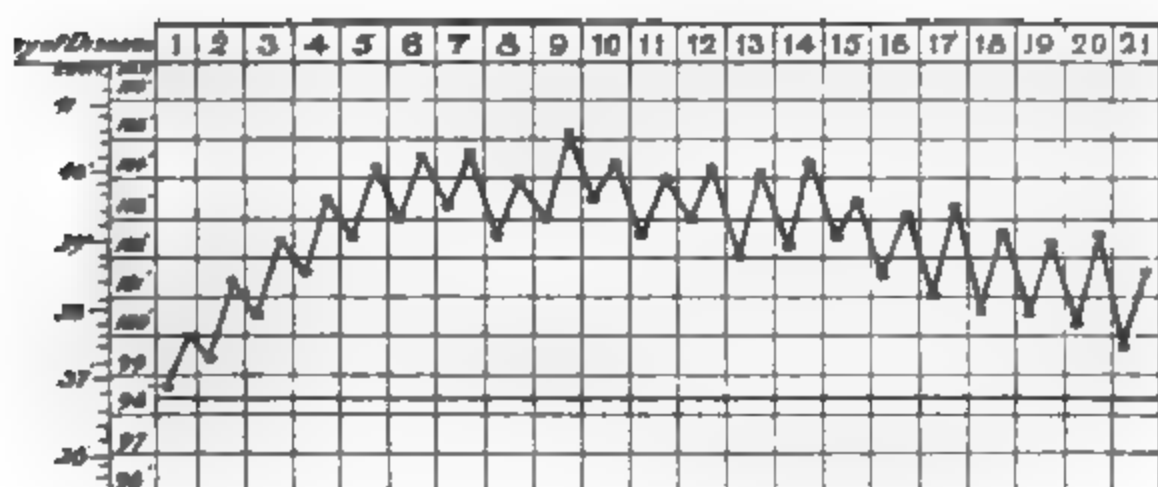


DIAGRAM. 1.—Temperature Chart of Typhoid Fever (uncomplicated case).

occurring in the Peyer's patches, the tongue is dry, especially the centre, and may be covered with sordes; the face is pale, the red, flushed cheeks; and although the patient is dull and listless, the eyes are brighter than in typhus. Diarrhoea is not infrequent, and if it has appeared already, it now comes much more severe, with abdominal distension and tenderness. The stools are pea-soup-like, alkaline, have a terrible smell, and contain much debris, altered blood, and, of course, many bacilli. There are certainly cases of typhoid in which diarrhoea is entirely absent, but should necrotic changes occur in the patches, it is common to find the typical stools.

Death is not uncommon during this week, and hæmorrhage and perforation are likely to be met with during the stage of moulting, although more probably they may occur during the following week.

During the *third week*, the clinical features depend on the

amount of ulceration present. There may be much diarrhoea, great meteorism and gurgling in the right iliac region, but in a favourable case the temperature should have begun to fall, and the physician may be only called upon to combat the progressive weakness following a protracted, continued fever. Great abdominal distension commonly precedes either hæmorrhage or perforation. Hæmorrhage occurs in 3 to 5 per cent of all cases, and may be said to be the cause of death in about one-twentieth of those proving fatal. It is generally preceded by a feeling of faintness; a marked fall of temperature and death may result even before the blood has appeared

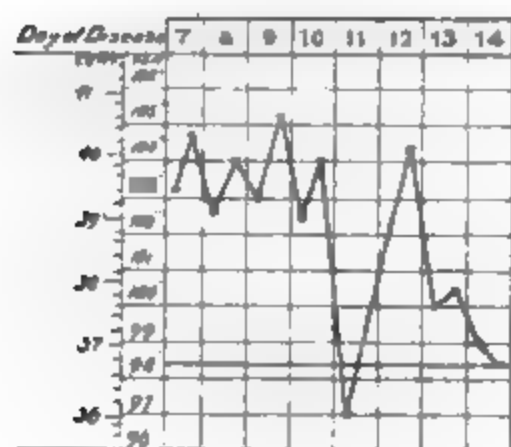


DIAGRAM 2. - Temperature Chart of Typhoid Fever showing sudden fall of temperature due to a severe hæmorrhage.

in the stools. Perforation occurs in from 2 to 3 per cent of all cases, and is the cause of death in 25 to 35 per cent of those which are fatal.

Abdominal pain is present as a typical feature of the disease, but it may become specially severe as the result of great meteorism, inflammation of the mesenteric glands, and, of course, peritonitis.

In the *Alimentary System* there is frequently a considerable amount of nausea and vomiting, and sometimes inflammation of the tonsils. Cholecystitis, due to infection of the gall-bladder, is by no means uncommon, and pain over that organ is a suggestive clinical feature.

In the *Hæmopoietic System* there is practically never a leucocytosis in uncomplicated cases, but the red blood corpuscles and hæmoglobin are diminished.

In the *Circulatory System* the first sound of the heart is almost invariably feeble, and the pulse full, soft, and dicrotic, but these are common results of any long-continued fever process.

In the *Respiratory System* there may be several complications, such as pleurisy, lobar pneumonia, and hypostatic congestion, while bronchitis and epistaxis have been already noted as among the early symptoms.

In the *Integumentary System* there are occasionally other

rashes present, generally of an erythematous type, while a cadaveric odour is certainly not uncommon in cases which are about to terminate fatally. Profuse sweating occurs once the fever has begun to abate.

In the *Urinary System* the presence of albumin is almost constant, associated probably with the excretion of both toxins and organisms by the kidney.

In the *Nervous System* headache and slight nocturnal delirium are the only symptoms in a mild case of typhoid fever. In severe cases there is a type of the fever to which the term nervous is applied. In it there are coma vigil, constant muttering delirium, subsultus tendinum, and plucking at the bedclothes, often terminating in profound coma and death. Hyperpyrexia, nearly always associated with marked delirium, is a grave danger in this type of typhoid fever. Peripheral neuritis, sometimes localised neuritis, or pain in connection with the muscles, may be associated with the disease, and more rarely a limited myelitis or other lesion of the cerebro-spinal system.

During the *fourth week*, in a mild case, convalescence begins, and after a varying period of time the patient completely recovers.

It must be remembered that many cases do not run a typical course; resolution may commence in the Peyer's patches without any necrosis occurring; in many cases profound changes may develop in the intestine without giving rise to the usual clinical features, and serious relapses are by no means uncommon even in a mild case, although generally as the result of dietetic error. We must also remember that besides the local action of the bacilli on certain tissues, there may be a pronounced and very generalised action of the toxins on the body as a whole, and a typhoid septicæmia is by no means rare.

Perhaps one of the most serious developments is relapse, which may set in at almost any period of the disease, and after one, two, or three weeks of apyrexia. The temperature may once more rise with a return of the typical intestinal and other symptoms. It should also be remembered, however, that a trivial error in diet may cause a slight and evanescent rise of temperature. The more serious relapses are almost certainly the result of re-infection, either of the same or of fresh Peyer's patches.

Types.—In a disease with such varied and complex groupings of clinical features many types may be described. Of these the most important are:—

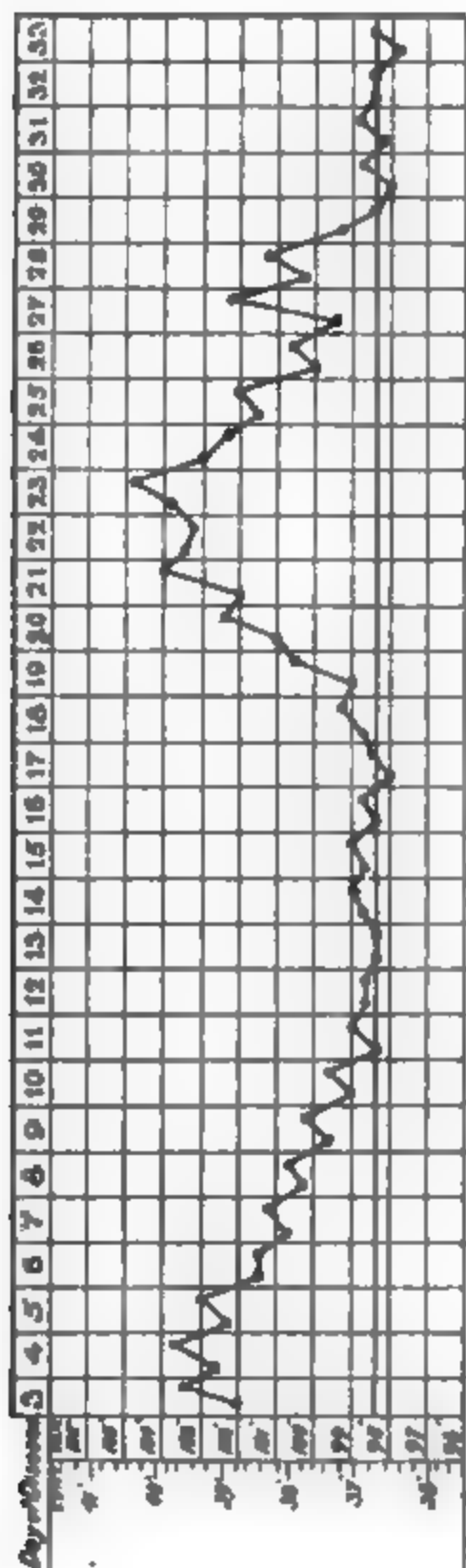


Diagram 3. Temperature Chart of a case of Typhoid Fever with remission about twentieth day.

(1) *A mild abortive type*, in which probably resolution occurs in or soon after the first week. The rash may be present, but the temperature falls with profuse sweating, and restoration to health speedily follows.

(2) *The ordinary type* of typhoid fever already described.

(3) *A gastro-intestinal type*, in which vomiting and diarrhoea are specially marked.

(4) *A malignant type*, in which, as a rule, the nervous symptoms are severe, or where death occurs prior to the definite development of the disease. So-called hemorrhagic typhoid, by which is meant hemorrhages from mucous surfaces, is a rare form.

(5) *The ambulatory or latent type* is perhaps one of the most serious forms, because the patient risks his life by carrying on his daily work in ignorance of the disease from which he is suffering, and may, by a dietetic error or over-exertion, readily cause the development of grave symptoms, such as perforation or hemorrhage.

(6) *Typhoid septicaemia* is the type already referred to, in which there is a general infection, often with no typical intestinal symptoms.

Complications.—The chief complications of the *Alimentary System* have already been described.

In the *Circulatory System* it should be kept in mind that the heart frequently is affected by the prolonged fever process. During the stage of convalescence thrombosis of the femoral veins, causing "white leg," may occur: the risk of separation of a portion of the clot must in such cases be guarded against.

In the *Respiratory System* lobar pneumonia, pleurisy, oedema of the lungs, and hypostatic congestion are of frequent occurrence.

In the *Integumentary System* the likelihood of bedsores, especially in asthenic patients, should be kept in mind, and crops of boils sometimes annoy the patient during convalescence.

In the *Urinary System* there is a risk of acute nephritis, while retention of the urine in an unconscious patient may produce cystitis.

In the *Nervous System* most of the more important complications have already been mentioned, but the possibility of inflammation of the eye or ear, although rare, should not be forgotten.

Diagnosis.—Perhaps no fever presented such difficulties of diagnosis prior to the introduction of the test introduced by Widal in 1896.

1. *The Widal Reaction.*—A culture of typhoid bacilli of less than twenty-four hours' age is added to a drop of sterile water. A little blood is taken from the finger or ear of the patient, and the serum diluted thirty times with distilled water. A drop of the diluted blood-serum is added to a drop of the typhoid culture in water, and examined under the microscope. In the event of the case being one of typhoid the bacilli soon cease to move actively, run into clumps (which is called agglutination), and this should occur certainly within an hour.¹ The reaction should be present after the end of the first week, and only fails in a small percentage of cases of typhoid. A similar reaction can be obtained in cholera, dysentery, glanders, and some other fevers by using a culture of the respective organisms in the same way.

2. *Isolation of Bacillus.*—In addition to the Widal test, one may endeavour to isolate the bacillus of Eberth from the stools, urine, or, best of all, from the spleen; but in many cases, notwithstanding the utmost care, the result is unsatisfactory.

3. *The Diazo-Reaction of the Urine*, described first by

¹ The agglutination test can also be applied by a naked-eye method, suspensions of dead typhoid bacilli being used with blood-serum from the patient.

Ehrlich, has been largely replaced by Widal's serum-reaction. The test consists in the following: Two solutions are prepared, (1) a saturated solution of sulphanilic acid in 5 per cent hydrochloric acid in water, and (2) a $\frac{1}{2}$ per cent solution of sodium nitrite in water. To a few c.c. of the urine in a test-tube add equal parts of solution (1), and a few drops of solution (2). Shake the test-tube, and add enough ammonia to render the mixture alkaline. When sulphanilic acid is acted on by nitrous acid, a compound is formed which unites with aromatic compounds found in certain urines, and amongst others the urine of a typhoid patient, to form a brownish-red colour, the froth being specially tinged. The test, although commonly positive in typhoid fever about the second or third week, is also present in cases of measles, in rapid tuberculosis, and in some other conditions.

These tests, together with the typical temperature chart, the presence of the rash, should it occur, and the enlargement of the spleen, are generally sufficient to prevent mistake; but the physician is more apt to consider his patient to be suffering from such diseases as *tubercular meningitis*, *acute miliary tuberculosis*, *lobar pneumonia*, *catarrhal pneumonia*, or *influenza*, and to ignore the possibility of typhoid altogether. It is a wise precaution to have the Widal reaction carried out in all doubtful cases, and more especially if the patient presents symptoms not in keeping with the original diagnosis. We have seen several cases diagnosed as catarrhal pneumonia or capillary bronchitis which turned out to be typhoid.

Prognosis.—The death-rate is rarely over 20 per cent. and generally only about 10 per cent: but the age, constitution, and habits of the patient greatly affect the chances of a satisfactory result, while it must be remembered that epidemics vary in their severity. Unfavourable conditions, such as excessive meteorism, hæmorrhage, perforation, or marked nervous symptoms, should make the physician cautious, while older patients (after middle life), alcoholics, very debilitated subjects, and extremely stout persons run a much greater risk.

Treatment.—*General.*—The patient should be placed in a well-ventilated sick-room, and the most absolute cleanliness is requisite, especially for the sake of those in attendance. A careful note of every little detail is of great value, such as a three- or four-hourly temperature chart; the times of feeding



the patient, with the amount taken; the number of hours of sleep; the quantity of urine; and the number of stools passed. From the very commencement the condition of the skin should be thought of. Cleanliness is essential, and the local use of a non-irritating antiseptic, such as boracic acid, will be found of much value, while a water-bed is a great comfort in most instances. On no account should the patient be allowed to get up for the evacuation of bowels or bladder, and in severe cases order cotton-waste to be used in place of the bed-pan. The patient should be kept absolutely at rest, free from excitement, and encouraged to sleep as much as possible.

Diet.—There is much difference of opinion as to whether the patient should order milk, perhaps peptonised, with egg-flip and similar easily digested and nourishing foods, or whether the patient should be starved, strength being merely kept up by a little iced alcohol and water with the minimum of other nourishment. The principle governing the dietetic régime is to have as little waste matter reaching the inflamed portion of the intestine as possible, and probably a middle course is advisable. A careful study of the stools should be made so as to find out whether any undigested food is present, and therefore causing irritation, and should we find no evidence of undigested food, a slightly more generous dietary may be cautiously instituted.

Special Symptoms.—*Pyrexia and Hyperpyrexia.*—Many physicians, and especially Germans and Americans, have used the bath system of treatment with great benefit. This consists in immersing the patient in water about 70° to 75° Fahrenheit, and either pouring cold water over him or applying cold-water compresses to the chest and abdomen. The patient remains in the bath about fifteen minutes, during which he usually becomes extremely blue and shivers with the cold. The baths are repeated every two or three hours, if necessary. As many as 200 baths have been given to a patient during an attack of typhoid fever. After each bath the patient is rubbed dry, and, if somewhat collapsed, a little alcohol is administered. This means continued pyrexia is prevented, and the death-rate in serious cases has certainly been considerably reduced.

For hyperpyrexia similar treatment may be adopted, or any or other of the methods described in the treatment of fevers generally (p. 5) may be preferred.

Diarrhœa.—Where diarrhœa is profuse and tending to be excessive, the dietary of the patient should be carefully revised, and either bismuth subnitrate (gr. 30-40), with Dover's powder (gr. 5-15), or small doses of lead acetate (gr. 2) given at frequent intervals. A starch and opium enema is often invaluable, and morphia suppositories (gr. $\frac{1}{2}$ -1) may be administered with great benefit. Not infrequently alteration of diet, and especially the substitution of boiled milk, relieves the patient.

Meteorism and abdominal pain are often relieved by the application of either hot fomentations or cold compresses over the abdomen, and sometimes by the use of such counter-irritants as turpentine stupes. Tympanitis is frequently prevented by the administration of intestinal antiseptics, such as salol (gr. 20), wood charcoal (gr. 30-60), or β -naphthol. An enema containing turpentine or asafœtida is often of service, and in very severe cases, where there is paralysis of the bowel, the introduction of a long, flexible rubber tube has given much-needed relief by drawing off some of the gas.

Hæmorrhage from the bowel is very frequently the result of excessive meteorism. Absolute rest should be enjoined, the quantity of food diminished, and replaced by pieces of ice to suck, while full doses of acetate of lead with opium or 10 minim capsules of turpentine should be promptly administered. An ice-bag should be applied to the abdomen; very generally alcohol or other stimulant may have to be administered owing to collapse.

Perforation usually results from one or possibly more ulcers, and an attempt should invariably be made to treat the condition surgically.

Peritonitis is relieved by full doses of opium administered hypodermically, and by the application of either warm poultices and fomentations or an ice-bag. In some cases operative interference may yield a faint hope of relief.

Heart failure should be prevented by careful attention to the heart-sounds, and the early use of digitalis, strychnine, and certainly diffusible stimulants.

*Bedsore*s can usually be obviated by the attention to cleanliness previously recommended, while a water-bed and the process of hardening the skin referred to on p. 6 are of special value in a long-continued fever like typhoid.

Headache is sometimes so severe that it may call for special treatment, when the use of the ice-bag or the internal administration of phenacetin in 10-grain doses every two hours generally proves sufficient. The patient should be kept absolutely quiet, and the room darkened.

Insomnia is troublesome in many fevers, and especially in phoid. Sulphonal (gr. 15-30), chloralamide (gr. 15-30), paraldehyde (m 60-120) are amongst the best hypnotics for a case of the kind.

Delirium is frequently associated with hyperpyrexia, though it may indicate that some grave nervous, respiratory, or other complication is present. Ice-bags applied to the head and the treatment for hyperpyrexia are invaluable.

PROPHYLAXIS.—It is hardly necessary to refer to the importance of pure and uncontaminated drinking water, nor is it requisite to do more than mention the risks of sewage getting into wells. The stools, probably also the urine, and most assuredly the sheets from the patient's bed, should be disinfected, either by using carbolic acid of a strength of 1 in 20 parts, or corrosive sublimate of 1 in 1000, and the greatest attention and care should be paid by the nurse to the careful disinfection of her hands.

Inoculation against Typhoid.—Wright's prophylactic is a fluid containing dead typhoid bacilli, and the specific toxin which has been elaborated in the course of cultivation in 1 per cent peptone bouillon. Flasks are incubated at 37°C for 14-20 days, then by raising the temperature to 40° to 60° Centigrade the bacilli are killed. Standardise the fluid and add a preservative, and give from 0.5 to 1.5 c.c. of each of the two inoculations. Little can be said as yet as regards the success of this treatment, while it usually causes considerable discomfort to the patients, both malaise and heightened temperature being present for several days. The mortality from typhoid after inoculation appears, however, to be lower, and the test of experience seems to indicate that the measure is of decided value.

PARATYPHOID

This remarkable fever closely resembles typhoid, but is entirely due to a distinct organism not very dissimilar to

the colon bacillus. The fever process is like a mild attack of true typhoid with splenic enlargement, a rose-coloured rash, and evidences of intestinal involvement. The chief points of difference are—1, that the essential lesion of typhoid in the Peyer's patches and solitary glands in the intestine is generally absent: (2) that a pathological condition more nearly resembling dysentery may be present: and (3) that the typical Widal's reaction is not obtained.

III. TYPHUS FEVER

(SPOTTED FEVER)

AN acute infectious fever occurring in epidemics, and due largely to dirt and insanitary conditions.

Etiology.—Several organisms have been described, and amongst others a diplococcus was isolated by Balfour and Porter during the last severe epidemic in Edinburgh. Dirt, overcrowding, starvation, and probably fatigue of mind and body, are all predisposing factors. Improved sanitary arrangements have tended to stamp out this very fatal fever. It may be transmitted by contact and by the clothes of infected persons.

Pathological Anatomy.—The spleen is invariably congested and enlarged. The blood is dark, but there is no distinctive pathological lesion.

Clinical Features.—The stage of incubation is generally about 12 days although sometimes it may be much shorter. The stage of invasion usually commences with a marked rigor, the temperature rapidly rises, the pulse is full, the tongue furred and dry, and there is frequently pain in the back, limbs, and head. The face is flushed, the conjunctivæ are injected, and there may be mild delirium at night.

The stage of *Exanthema* appears from the 3rd to the 5th day. The rash comes out first on the trunk, on the backs of the hands and elbows, and later, and to a less extent, on the face. It is a dusky mottled eruption, often described as of mulberry appearance, and at an early period little petechial hæmorrhages develop in certain of the spots. The temperature remains high, from 103° to 105° F., the tongue becomes brown

and dry, the urine is scanty, with a heavy uratic deposit and often a little albumin; the apathetic appearance of the patient is very typical. There is almost always a certain amount of delirium, especially at night, and dulness of hearing, which necessitates loud speaking when attempting to communicate with the patient. In a severe case the face becomes dusky, and coma vigil and subsultus tendinum supervene.

There is a distinctive odour about the body of a typhus patient, which is certainly very unpleasant and is said to be of diagnostic value.

The termination by *crisis* generally begins before the end of the second week, the temperature usually falling to normal

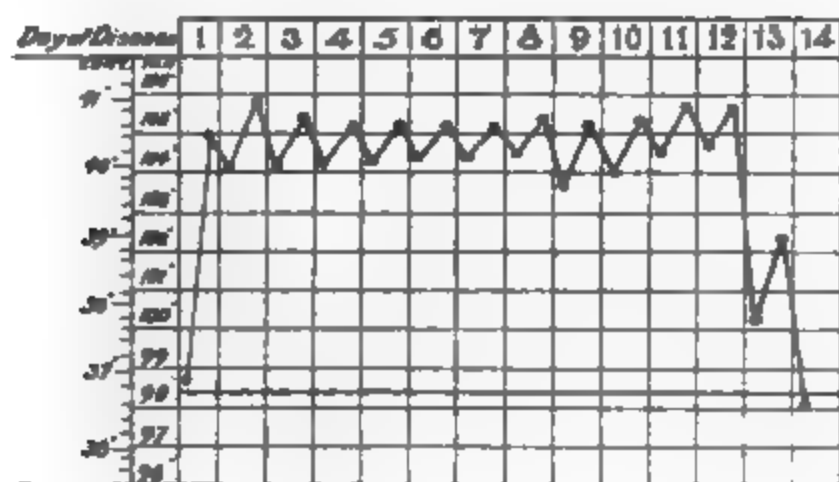


DIAGRAM 4. - Temperature Chart. Typhus Fever Recovery

within twenty-four hours, and the patient at once becomes brighter and less apathetic.

Complications.—Perhaps the most fatal complication is perpyrexia, which may cause death at an early period in the fever. Heart failure, pulmonary complications, especially poststatic congestion, gastro-intestinal catarrh, Bright's disease, a deep coma or wild delirium, may occur. Occasionally lebitis, sometimes paralysis (hemiplegia or peripheral neuritis) and even insanity, follow the fever.

It is hardly necessary to state that a malignant type of typhus is common.

Diagnosis.—The petechial rash, the delirium, and the continued high temperature rarely make a mistake possible. *Scorbutus* is not as a rule associated with an elevated temperature, while *flea-bites* show a marked puncture which can be easily seen with a magnifying-glass. In *measles* it is rare to find a hæmorrhagic rash, and in *uræmia* there should be no

rash at all. It is in isolated cases that the physician's skill is likely to be taxed.

Prognosis.—The death-rate varies considerably. The older the patient the higher it becomes. It is at least 10 per cent. Alcoholism, the appearance of marked nervous symptoms and pulmonary or cardiac complications render the prognosis grave.

Treatment.—The patient should be isolated along with those in attendance on him. Attention should be paid to the bladder, and as a rule the patient's skin should be sponged twice a day with some agreeable antiseptic lotion. The food is most important; a generous though simple diet must be ordered, and nourishment administered every two or three hours. Chloral or the bromides should be given for delirium, the bowels should be attended to, and the headache relieved by the administration of phenacetin or similar remedy.

The PROPHYLAXIS of the disease is of enormous importance. Sanitary improvements, attention to prevent overcrowding, and the careful separation of infected cases, with subsequent disinfection of the houses from which the patients have come, have done much to stamp out the disease from our large towns.

IV. SCARLET FEVER

(SCARLATINA)

AN acute infectious fever characterised by a brief stage of invasion, with sore throat, high temperature, and a typical rash.

Etiology.—It occurs generally in epidemics, but sporadic cases frequently crop up where epidemics have previously existed. It is most common in childhood, rare in infancy, but adults do not escape. Females appear to suffer more than males. Desquamation is probably the period of greatest infection, the scales of skin being infective. Milk is often the medium of infection.

Klein has isolated a streptococcus from the blood of scarlet fever patients, which he believes to be the specific organism. It grows rapidly on gelatine and other media, forming whitish

colonies, and it seems highly probable that this organism is the genuine streptococcus of scarlet fever.

Pathological Anatomy.—It is remarkable how rapidly the swelling of the skin disappears after death. There are no constant changes, but the throat inflammation with tonsillar suppuration is usually present, with sometimes acute inflammatory oedema of the neck, acute suppuration of the neighbouring lymphatic glands, or occasionally a burrowing cellulitis. The spleen is often enlarged and soft. There may be endocarditis and pericarditis, and not infrequently there is evidence of pulmonary complications, such as bronchitis, pneumonia, pleurisy, and hypostatic congestion, while in not a few cases of scarlet fever acute nephritis is well marked at death. In the nervous system perforation of the tympanic membrane,

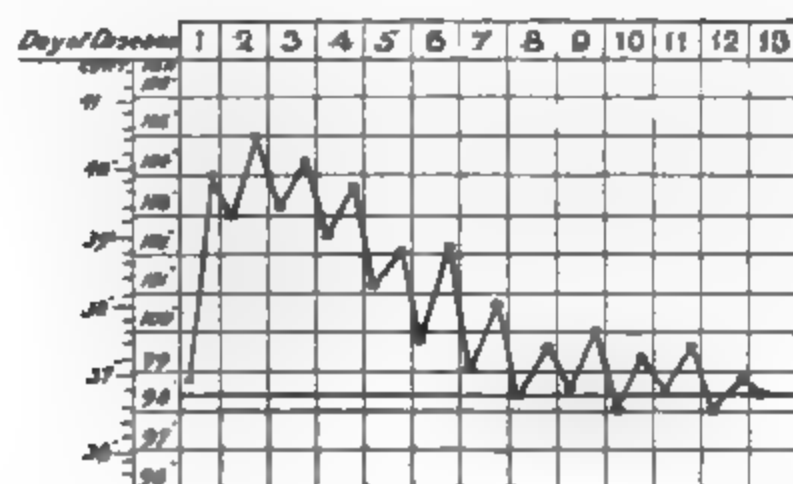


DIAGRAM 5.—Temperature Chart. Scarlet Fever Case terminating favourably

the result of inflammation of the middle ear, is extremely common, and may lead to mastoid abscess, septic meningitis, and sometimes abscess in the brain. In the locomotory system the joints are often enlarged and may suppurate.

Clinical Features.—The stage of *Incubation* is 1 to 8 or 9 days, with no symptoms.

The stage of *Invasion* is often about a day, with malaise, shivering, headache, sore throat, and generally a rapid rise of temperature (102°-104° F.) and pulse, and in children it frequently sets in with a rigor. On examination the throat is found to be red, the tonsils and fauces being swollen and inflamed.

The stage of *Eruption*. Generally on the second day a bright red, punctiform spots appear, first on the neck and chest, and then rapidly spreading over the body. The chin,

an area round the mouth, and the scalp usually escape. The whole skin is early hyperemic, and later bright scarlet. Sometimes there are small papular elevations, occasionally sudamina, and in malignant cases petechial

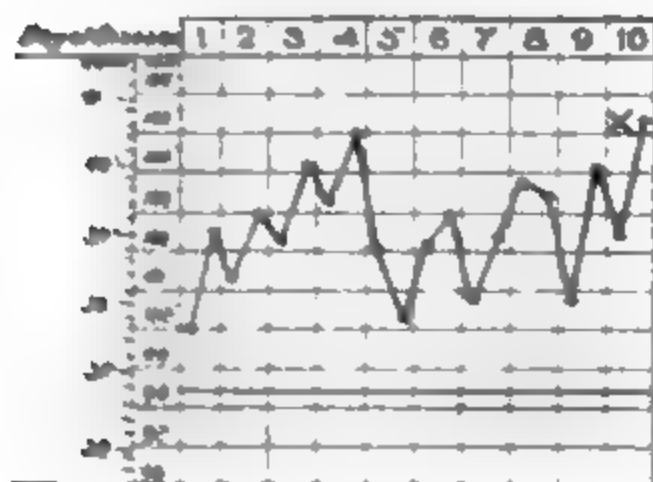


FIGURE 1. Temperature chart. Scarlet fever. (From a case of scarlet fever with both a tooth and a skin eruption.)

hemorrhages. The skin, especially in the neighbourhood of the joints, is so markedly swollen as to feel stiff and make the bending of the fingers difficult. While the face swells slightly, there is as a rule comparatively little rash. Pressure with the finger leaves a white mark for some seconds on the skin.

The tongue is at first covered with a whitish fur through which the hyperemic fungiform papillae shine like bright red spots dotted over its surface. This is the white strawberry tongue. When the fur is shed and the tongue becomes bright red with the fungiform papillae still showing prominently, it forms the red strawberry tongue. For a long time the patient's tongue remains typical.

During the stage of convalescence the throat symptoms continue, there is pain on swallowing, the voice is altered, and the tonsils are markedly prominent, while very frequently one or both of them suppurate. The temperature remains high, about 101° F., until the rash begins to fade, which it does about the 10th day. A convalescent temperature is usually present.

The skin eruption is generally by now

well advanced, and the hyperemia has become more marked. The eruption is now beginning to be more extensive, and the skin is more swollen. There may be some small papular elevations, and the hyperemia is more marked. The temperature remains high, about 101° F., until the rash begins to fade, which it does about the 10th day. A convalescent temperature is usually present.

Types.—*Scarlatina Simplex*.—Eruption with no throat symptoms.

Scarlatina Anginosa.—Eruption with more or less severe throat inflammation. Where the throat suffers, the temperature rises more markedly, and even in a favourable case it falls very gradually.

Scarlatina Latens.—Where there is no eruption, but desquamation, or possibly the development of sequelæ, indicates the existence of the disease.

Scarlatina Maligna.—Severe malignant types of the fever occur with either (1) marked nervous phenomena, and especially coma vigil and subsultus tendinum, sometimes called the ataxic form; or (2) a hæmorrhagic form, with petechial and other hæmorrhages. Both these types are generally fatal at an early stage.

Complications and Sequelæ.—In the *Alimentary System* there may be extensive phlegmonous inflammation of the tissues in the neighbourhood of the pharynx, and rarely there is marked gastro-intestinal catarrh.

In the *Hæmopoietic System* the lymphatic glands of the neck may suffer as already indicated.

In the *Circulatory System* endocarditis, sometimes ulcerative, and pericarditis may be present, while myocarditis is by no means uncommon.

In the *Respiratory System* pleurisy with empyema is probably the most frequent complication, and generally occurs in children.

In the *Integumentary System* bed-sores may form on points of pressure.

In the *Urinary System* an acute parenchymatous nephritis is by far the most important complication; it generally appears during the second or third week, and not infrequently is the result of exposure to cold, and may follow in even the mildest case. There is little œdema, but suppression or the passage of small quantities of highly albuminous urine with much blood, and many blood and epithelial casts, indicate the onset of the attack. In every case of scarlet fever a careful examination of the urine should be made at short intervals until the patient is fully restored to health.

In the *Nervous System* the most important complication of scarlet fever is middle-ear suppuration, the infection spreading

up the Eustachian tube, and resulting often in perforation of the tympanic membrane.

In the *Locomotory System* the joints may swell, become extremely painful, and sometimes suppurate.

Diagnosis.—In *measles* the longer period of invasion extending to 3 or 4 days, the typical crescentic rash, and the presence of the marked coryzal phenomena, with the absence of the general hyperæmia of the skin, should enable a definite diagnosis to be made. In *German measles* the fever is usually of a milder type; the rash may closely correspond, but is less extensive, and the cutaneous hyperæmia is not so marked; further, the glandular enlargement in German measles is usually out of proportion to the involvement of the throat.

In *scarlatinaform erythema* there may be a somewhat typical rash with resultant desquamation, but the absence of marked temperature and throat symptoms helps the diagnosis.

In *acute exfoliative dermatitis* there are, as a rule, no throat symptoms at all, while the desquamation is by far the most distinctive feature of the disease. *Diphtheria*, or diphtheroid inflammation, is not infrequently a sequel to scarlet fever, but the examination of a swab from the throat prevents any mistake being made. Certain *drug rashes* also resemble the eruption of scarlet fever, such as those due to belladonna, quinine, and salicylate of soda, but with a little care there should be no room for doubt.

Prognosis.—The milder cases in recovery, but it must be remembered that the death-rate is often as high as 15 per cent, a fatal result being frequently present in young children under 5 years of age: *scarlatina maligna* is generally fatal. Hyperpyrexia, early collapse, marked vomiting and diarrhœa, prominent nervous phenomena, and a severe attack of scarlatinal nephritis, are all grave features.

Treatment.—An ordinary fever treatment should be sufficient for a mild case, but the most rigid precautions must be taken to prevent the spread of the disease, and, if possible, patients should be sent to a fever hospital. Failing this, the sick-room selected should be large and airy; unnecessary furniture should be removed, along with all curtains, carpets, etc. The diet should consist largely of milk and beef-tea, while aerated waters and fruits, such as oranges and lemons, may be freely permitted. During the period of **pyrexia**,

sponging the patient is most refreshing; and when desquamation has commenced, the skin should be rubbed over with weak carbolic oil (2-5 per cent), carbolated vaseline of a similar strength, or thymol in vaseline (gr. 10 to the oz.). The patient should be warmly clad, and especially so if allowed up during the period of desquamation, remembering the risk of renal complications. The urine must be examined for albumin, as already mentioned, every few days, and similar care is paid to the ears, especially in cases in which the throat is severely affected.

For the throat symptoms much benefit may be obtained by the application of the *glycerinum acidi carbolici* (1 part of 2 or 3 of glycerine); or solutions of chlorate of potash, permanganate of potash, and other antiseptics may be used for gargling. Cold compresses applied externally are useful, and the internal administration of quinine, tincture of the perchloride of iron, guaiac, salicylate of soda, and other remedies, is of great value.

Hyperpyrexia should be treated by sponging, the cold bath, and the other methods mentioned on page 5, taking care to remember the frequent necessity for stimulation, as the temperature has been reduced. If the middle ear suppurates, it may be necessary to incise the tympanic membrane.

Other complications which may arise should be appropriately treated.

Serum therapy has been tried, but the results obtained so far are inconclusive.

PROPHYLAXIS.—Probably the patient is most infectious during desquamation, and therefore attention to the smearing of skin with an antiseptic, and the subsequent use of baths to separate the scales of skin, are invariably insisted on. On consideration should a scarlet fever patient be considered infected until a period of at least six weeks has elapsed since the rash came out, and every care must be taken to infect the clothes and anything which has been used in the room.

V. MEASLES

(MORBILLI; RUBEOLA)

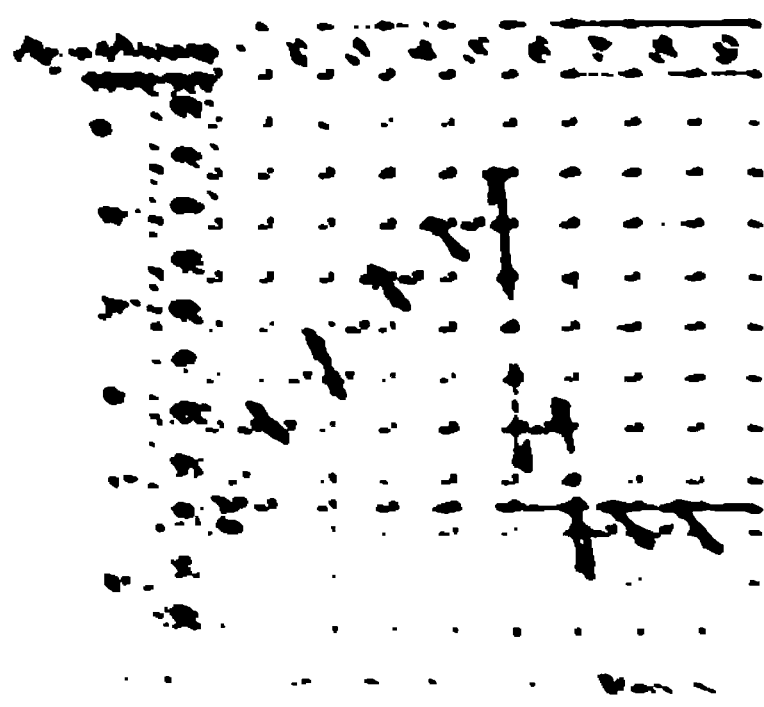
A CONTAGIOUS fever, characterised by an eruption of typical spots, and associated with catarrhal inflammation of mucous membranes, especially the eyes, nose, and respiratory tract.

Etiology. It spreads in epidemics, which are rarely long absent from large towns, and especially attacks children, although it may occur at all ages. It is most prevalent in spring and autumn. One attack protects from a second, as a rule. It is probably most infectious during the stage of eruption.

Pathological Anatomy.—There is a specific inflammation of the mucous membranes of the respiratory and, to a less extent, of the intestinal tracts, but the organism is unknown.

There is an oedema of the cutis, to which, probably, the typical measles papule is due.

Clinical Features.—The stage of *Incubation* of about 10 days is followed by the stage of *Prodrom*, lasting 3 or 4 days. During this latter stage the temperature gradually rises, there is quickened pulse, malaise, and the typical nasal catarrh with conjunctival congestion and more or less



itchiness. The temperature may slightly fall before the next stage appears. The stage of *Eruption* lasts 3 or 4 days, and is characterised by raised red spots, which appear first on the face and spread to the rest of the body. The eruption is accompanied by conjunctival congestion and more or less

itchiness. The temperature may slightly fall before the next stage appears. The stage of *Eruption* lasts 3 or 4 days, and is characterised by raised red spots, which appear first on the face and spread to the rest of the body. The eruption is accompanied by conjunctival congestion and more or less

are found on the buccal mucosa in a large proportion of cases. They appear very early, and are therefore of diagnostic value. Later these spots coalesce, but many bluish-white specks can still be seen. The general rash fades on the 6th or 7th day, when the temperature rapidly falls by *crisis*, and desquamation begins about the 8th day in the form of fine branny scales, very different from the large flakes of scarlatina. The stage of *convalescence* is rapid, if there are no respiratory complications.

Special Types.—The *Hæmorrhagic Type* of measles, with purpuric rash and hæmorrhage from the mucous membranes, sometimes met with, and the so-called *Typhoid Type* is applicable to cases of measles which show extreme prostration, where an excessive dose of the poison is threatening the life of the patient.

Complications.—Capillary bronchitis leading to catarrhal pneumonia is to be dreaded, especially in delicate children, and frequently causes death. Eye and ear inflammation may occur, while cancrum oris is a rare sequel in delicate children. Persistent gastric or intestinal catarrh is not infrequent, and the disease may lead to permanent ill-health.

Diagnosis.—From *scarlet fever*, the typical coryza, the rash, and the long period of invasion are absolutely diagnostic. From *German measles* the rash and the absence of the sore throat and the marked enlargement of the lymphatic glands are at least suggestive. In *typhus* there is always delirium, the face is little affected by the rash, and the spleen is typically always enlarged. The initial *small-pox* rash may be confused, but rarely are the awful headache and back-ache of small-pox present in measles.

Prognosis.—Generally recovery from the fever, but the respiratory complications cause a large death-rate in young children. In certain epidemics the death-rate reaches a high percentage, but often a fatal result is due in part to the age of the child, the insanitary surroundings, and careless nursing.

Treatment.—A mild case requires practically no medicine, only ordinary dietetic and hygienic measures. Keep the child in a fairly warm room, give a mild fever mixture (R. spiritus ætheris nitrosi ℥ 15, and liquor ammonii acetatis ad ℥ 1), and feed on milk diet or beef-tea. Where there is much bronchitis a steam-kettle should be invariably used, and

the utmost care taken to avoid the risk of extension downwards to the lungs.

It ought to be remembered that measles is contagious, and at least a fortnight should elapse before members of the family coming in contact with the patient are free from the risk of infection.

VI. GERMAN MEASLES

(RUBELLA ; RÖTHELN)

AN infectious, eruptive fever possessing characteristics both of scarlet fever and measles.

Etiology.—There is much difference of opinion as to whether some cases are not scarlet fever and others measles, but our present position should be that it is due to a distinct organism. It is usually an affection of childhood, and one attack does not appear to confer any immunity, nor does an attack of this fever protect the patient from measles or scarlet fever. It is most commonly met with in epidemic form during spring-time.

Pathological Anatomy.—The lymphatic glands of the neck are especially enlarged.

Clinical Features.—The stage of *Incubation* is 5 to 14 days. The stage of *Invasion* commences with malaise, headache, pains in the back and legs, coryza, sore throat, and rise of temperature, while nausea is not infrequent.

Usually about the second day the stage of *Eruption* begins, the rash appearing first on the face and neck, or on the trunk, and rapidly spreading over the body. The spots are round or oval, discrete, or tending to coalesce, forming irregular patches somewhat simulating the crescentic rash of measles. At the same time the mucous membrane of the pharynx and, to a less extent, the nose and the conjunctivæ inflame. The typical enlargement of the lymphatic glands affects those in the neck, and especially the glands behind the sterno-mastoid muscle.

In 2 or 3 days the eruption fades, the temperature, which may have reached 102° F., falls by *crisis*, and during the stage of *Convalescence* desquamation may occur to a slight extent.

Types.—The disease is usually extremely mild, and the patient may never be confined to bed, but malignant cases occasionally occur in which the patient dies prior even to the age of eruption.

There are very few, if any, definite complications.

Diagnosis.—The rash certainly resembles both *scarlet fever* and *measles*. The stage of invasion is shorter than in measles, which ought to differentiate it from that disease: while the coryzal symptoms, which suggest measles, should prevent the diagnosis of scarlatina being made.

Prognosis.—General recovery; severe epidemics are rare.

Treatment.—Where there is pyrexia the patient should be kept in bed; otherwise an ordinary fever mixture is all that is requisite. The diet should be on the usual lines, at least for a few days of pyrexia.

VII. SMALL-POX

(VARIOLA)

acute contagious fever with a typical pustular eruption.

Etiology.—It is due to a specific organism as yet undiscovered, which causes wide-spread and general epidemics unless a community protected by vaccination.

The infection is conveyed by the secretion from the pustules, and certainly by the scales of skin during convalescence. A minute bacillus has been discovered by Klein, and he ascribes spore-like bodies which appear at a later date, while Henselmann has isolated a protozoon; but there is still much doubt as to the specific organism or organisms present. The infection of the child *in utero* has occurred. It is common at all ages, most fatal in the very young, and both sexes suffer equally. Certain races appear to have a higher death-rate from small-pox than others. The Mexicans, for example, died by thousands in one epidemic of small-pox.

Pathological Anatomy.—The typical papule is found in the superficial layers of the rete mucosum close to the cutis. The epithelial cells are granular and cedematous, and the processes are described by Unna—a reticulating degeneration and an epithelial oedema. The process advances most

rapidly at the periphery of the pock, causing a balloon-like appearance, the central part undergoing less-marked change, and thus producing the typical umbilication of the pock. There is always active inflammation round about the pock, and a rapid invasion of leucocytes into the affected area towards the end of the papular stage, when the pock becomes hard and shot-like. Scarring depends upon damage to the cutis vera by scratching or extension of suppuration downwards.

The spleen is enlarged. There is sometimes myocarditis, rarely endo- or pericarditis. Pocks may appear on the larynx, causing ulceration, while the bronchi may be involved, and even septic pneumonia result. The skin often shows intense congestion, and gangrene of the genitals may occur,

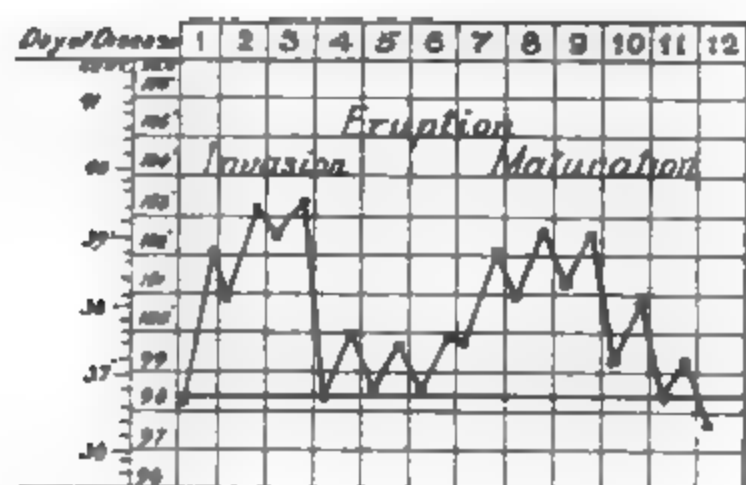


DIAGRAM 5 - Temperature Chart Small-pox Discrete case Recovery.

especially where the patient is suffering from a coincident severe gonorrhoea. Bright's disease may develop during the stage of convalescence, and sometimes suppuration of joints.

In a malignant case the patient dies before the eruption comes out, or there may be extensive hæmorrhage from serous membranes into the pocks or elsewhere.

Clinical Features. -The stage of *Incubation*, 11-14 days. The stage of *Invasion*, lasting from 2 to 3 days, is accompanied by very definite phenomena. There are rigors, severe frontal headache, pains in the back and epigastrium, vomiting, and a temperature which rapidly rises each day to a higher point than the preceding day, and with only a slight morning fall. There are sometimes convulsions in children. The initial rash, resembling scarlet fever or measles, and sometimes petechial, usually comes out on the lower region of the trunk, the inner aspect of the thighs, and the lateral regions of the chest.

These rashes are uncertain as regards appearance, extent, and duration. Sweating is often profuse.

In the stage of *Advance* or *Eruption*, at first the temperature falls and papules appear on the forehead, face, limbs, and trunk, and they may be seen on the soles of the feet and palmar aspect of hands and fingers. On the 2nd or 3rd day the papules are shot-like, become vesicular, are as a rule discrete and umbilicated, but may even at this period show a tendency to be confluent. By the 7th or 8th day the vesicles become pustules, and are surrounded by a zone of inflammation. This is termed *maturation*, and the temperature rises once more during this stage, as indicated in the chart. The pocks are tense and painful, there is much swelling of the skin, and the features of the face may almost be unrecognisable.

In the *confluent* form the pocks may be first discrete and then later run together; they usually appear early, often before the 4th day, and the initial discomfort is much greater. During the stage of maturation the inflammation is more intense, the temperature much higher (104°-105° F.), and delirium and other nervous phenomena are common. Diarrhoea at this time is very frequent, due largely to septic absorption.

When the pocks burst the patient becomes extremely moribund. About the 10th to the 18th day the pustules dry and form cicatrices, and soon afterwards desquamation begins. This process is often called *desiccation*, and the whole skin may be shed *en masse*.

Resolution is by *lysis*, and for long afterwards the skin remains red and unsightly. During the process of cicatrization the itching is apt to be intense.

Salivation, vomiting, and constipation, more often diarrhoea, may be present. Hypostatic congestion of the lungs, pneumonia, and pleurisy are not infrequent, and sometimes the diphtheria attacks the larynx. There may be albumin in the urine, and a degree of delirium is very common, while convulsus tendinum and coma vigil may be present in severe cases.

As already indicated, pocks may be found on the mucous membranes, and sometimes hæmorrhage occurs in connection with the pocks.

Types.—*Variola Discreta*, where the pocks are isolated, has already been described.

Variola Confluenta is much more serious, 50 per cent being fatal. The stage of maturation is very severe, and the pocks are specially confluent on the face and limbs. There is not the same remission of fever prior to maturation. The mucous membranes are more apt to be affected, and generally there

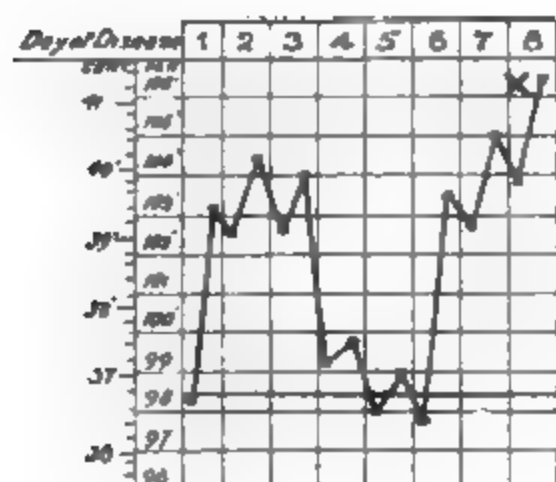


DIAGRAM 9. Temperature Chart Malignant Small-pox. Death on eighth day

are pocks on the conjunctiva or even the cornea. These may readily lead to panophthalmitis from perforation of the eyeball. The patient usually succumbs between the 10th and 28th days, and the constitutional phenomena are apt to be extremely severe. Vomiting, persistent diarrhoea, salivation, albuminuria, and various respiratory complications are present as a rule.

Variola Maligna.—In this type the patient may die from excessive doses of the poison prior to the stage of eruption, or excessive hæmorrhage may occur into the pocks or from the mucous membranes (stomach and bowels) and the kidneys, etc. Not infrequently the initial rash is hæmorrhagic. The hæmorrhagic form is called black small-pox.

Varioloid or modified Small-pox is small-pox occurring in a vaccinated person. There is a well-marked stage of invasion, but the rash comes out very rapidly. The pocks are few in number, are present on the face and limbs, and at an early period become pustular, implying that maturation is earlier than in the ordinary case of small-pox. Varioloid is just as infectious to others, and hence the importance of a correct diagnosis.

Complications.—*Alimentary System.*—The throat, tongue, and stomach may all be involved by the pocks. Gastric and intestinal catarrh is not uncommon, especially in children, and vomiting has been already referred to as a typical manifestation of the disease.

Hæmopoietic System.—There is a marked leucocytosis, especially during maturation, and the lymphatic glands are often enlarged.

Circulatory System.—Myocarditis, endocarditis, and pericarditis have been mentioned, and may occur as complications, while more frequently functional cardiac disturbances, especially palpitation, trouble the patient during convalescence.

Respiratory System.—Laryngitis, bronchitis, broncho-pneumonia, pleurisy, empyema, and hypostatic congestion are all frequent occurrences.

Integumentary System.—Erysipelas is not uncommon, and when gangrene may involve the genital organs should there be coincident gonorrhœa, while boils frequently occur during convalescence, due to interference with the sebaceous glands.

Urinary System.—Bright's disease occasionally supervenes.

Nervous System.—The pocks on the conjunctiva and cornea may cause blindness. Deafness, sometimes due to inflammation spreading up the Eustachian tube, may also be present. Delirium is common in severe cases, and coma, more rarely apoplexy, and paralyzes of different types may occur.

Locomotor System.—Inflammation of joints sometimes follows during desquamation, and may lead to permanent lameness.

Diagnosis.—The back-ache, headache, and the nocturnal sweating are typical. The shotty hardness of the pocks and their appearance under the horny skin of feet and the carpal aspect of fingers are also distinctive. Not merely may small-pox be differentiated by attention to these features, but the stage of invasion is preceded in that disease by no typical prodromata, and the rash comes out in 24 hours, usually in crops of spots on different days. The initial rash, measly or not fever-like, must not be forgotten, and the sites of the rash in small-pox should be carefully noted. Varioloid is mainly most apt to be confused with chicken-pox.

Prognosis.—In mild cases recovery is common, the mortality rate being low, and especially so in cases modified by vaccination. In confluent and hæmorrhagic small-pox at least 25 per cent die.

The following features are unfavourable:—

The quality and character of the eruption, especially hæmorrhagic.

The extent of mucous membrane involved in the eruption, if considerable.

(3) The age of the patient, the disease being particularly fatal in the very young.

(4) The marked nervous symptoms present, especially wild delirium, deep coma, or intractable sleeplessness.

(5) The previous habits of the patient; alcoholism is peculiarly unfavourable.

(6) Pregnancy nearly always leads to abortion or miscarriage, and so increases the risk to the patient.

(7) Insanitary surroundings, especially dirt, an insufficiency of fresh air, and unsatisfactory diet and nursing.

On the other hand, the presence of well-marked vaccination scars is a good feature, as it greatly minimises the risk of a severe type of the fever.

Treatment.—In a mild case comparatively little treatment is necessary, but in a severe attack the utmost care by both nurse and doctor may alone save life, obviate disfigurement, and prevent much suffering. The sick-room should be large and well ventilated, or, better still, a tent or wooden shed should be used, but the temperature must be kept up to about 50° or 60° F. A water-bed should be provided, and extremely light but warm clothing. The skin is unusually sensitive, and the finest texture of night-gown and bed-linen adds greatly to the comfort of the sufferer. The room should be kept darkened, or, better still, the windows may be covered with thin orange-coloured tissue-paper, so as to obstruct the actinic rays, to which the scarring is largely due. Carpets and curtains and all unnecessary furniture should be removed. The diet should consist of milk, soups, custards, jellies, and similar easily digested food. Relieve the back-ache with opium, and order ice and aerated water for the intense thirst and vomiting. During the stage of maturation feed generously, and, where possible, administer alcohol freely, because at that time the patient is exposed to the danger of septic absorption.

A warm bath soothes the itching, and the skin should be anointed with an antiseptic oil, the pustules pricked, especially those on the face, and cold water compresses used for allaying the discomfort. Watch the eyes carefully, washing frequently with boric lotion where pocks are present on, or near, the eyeball. Do not expose the patient to a bright light, and remember the necessity for abundance of fresh air for patient and attendants alike.

VIII. VACCINATION

(VACCINIA)

VACCINATION was discovered by Dr. Edward Jenner in 1798, when he vaccinated with cow-pox a boy eight years of age.

Prior to this the inoculation method was practised. Inoculation consisted in the introduction of small-pox matter direct from a small-pox vesicle prior to the pustule stage. The patient had, as a result, an attack of small-pox which was usually mild, but was naturally extremely infectious, and could cause infection to others of possibly a severe type of the disease. The death-rate proved, however, to be extremely small, but the patient required to be confined in hospital during the process.

Vaccinia, or cow-pox, is probably small-pox passed through a calf. The vesicles in cow-pox are found on the teats or udders of cows, and the disease is transmitted to the hands of those milking the cows, and also from one cow to the other by contact. Jenner noticed that in an epidemic of small-pox, milkmaids, who had previously become accidentally inoculated with vaccinia, had a marvellous immunity from small-pox.

As we, unfortunately, are still in doubt as to the specific character of small-pox, it would be futile to compare the characters of vaccinia and small-pox together.

Primary Vaccination.—*Local Phenomena.*—2nd to 3rd

day.—Slight papular elevation.

5th to 6th day.—Distinct vesicles of bluish-white colour, with raised edge and central depression.

7th to 8th day.—Areola forms round base, and spreads for 2 or 3 days. It is circular, 1 to 3 inches in diameter, and may be accompanied by hardness and swelling of subjacent tissues.

8th day.—Vesicle perfect, plump, distended with clear fluid, firm margin, with marked central cup.

10th to 13th day.—Areola fades and disappears.

14th to 15th day.—Scab forms as result of drying of vesicle. Scab contracts and blackens.

20th to 25th day.—Usually on 21st day scab falls off, leaving a cicatrix which is circular, depressed, and permanent.

Rise of Temperature.—Slight from 5th to 7th day, greater from 8th to 10th day, i.e. during areola stage.

The Complications which may result from vaccination depend largely upon three factors: *Firstly*, the care with which the trivial operation is performed; *Secondly*, the health of the child at the period of vaccination; and, *Thirdly*, whether calf lymph or humanised lymph is used.

To obviate complications the following precautions should be attended to:—

1. The site of vaccination should be carefully cleansed with an antiseptic lotion, then washed with boiled water, and the part covered after the operation with a pad of aseptic wool, and during the vesicle stage protected from injury.

2. The child should not be vaccinated unless it is in good health, and the period of teething should be, if possible, anticipated by previous vaccination.

3. Humanised lymph causes less reaction in the case of a delicate child, but the child from whom the lymph is taken must be healthy, and the possibilities of both tuberculosis and syphilis must be taken into account.

The chief complications of vaccination are the following:—

Ulceration and *suppuration* at the site of the operation, with abnormal enlargement of the lymph glands in the armpit. This is generally due to carelessness. *Eczema* may also be caused where a child has a predisposition to this affection. *Erysipelas* is not infrequent, and is due to want of aseptic precautions. *Excessive constitutional disturbance* is only marked in delicate children, and especially when calf lymph and not humanised lymph is used. It is wise to delay vaccination until the child is in at least fairly good health. *Syphilis* and *tuberculosis* are rarely inoculated, and only when humanised lymph is used and insufficient care taken in the selection of a healthy child as a medium.

Generalised vaccinia is rare. By this is meant the appearance of the typical spots over the body generally, although more numerous as a rule on the limb which has been vaccinated.

Revaccination should be performed every seven years, and invariably after exposure to the risk of small-pox. A good mark ought to be visible on the arm or leg, and should be looked for before protection from small-pox can be assured. As a rule four sites of inoculation are considered necessary, and the insertion of the deltoid muscle on the left arm is the usual position selected.

IX. CHICKEN-POX

(VARICELLA)

N acute infectious fever most common in early life, consisting in the appearance of successive crops of vesicles; it is quite distinct from small-pox.

Etiology.—Generally epidemic, sometimes sporadic. It is a disease of childhood or infancy, and no specific organism has yet been discovered. It is very contagious, but there is doubt as to the possibility of inoculation.

Pathological Anatomy.—Unna describes reticulative degeneration, and to a certain extent an œdematous change in the epithelial cells in the pock, resembling, but in a milder degree, what occurs in a small-pox pustule. The vesicles are generally simple and not umbilicated.

Clinical Features.—Stage of *Incubation*, 10 to 15 days. Stage of *Invasion*, with slight fever and malaise, is followed in a day by the stage of *Eruption*. Red, raised papules appear first on the trunk, forehead, and face, which in a few hours become vesicles containing a clear fluid. They may be surrounded by a slight area of hyperæmia. In one to two days the fluid becomes cloudy or even purulent, and soon dries up, leaving no scar. Several crops appear on successive days, often amounting to two to three hundred spots in all, and are spread more or less over the whole body. There is slight feverishness with eruption, and specially so where much pustulation, or where a tendency to a confluent form occurs.

Diagnosis.—Differentiate from *small-pox* by the more superficial, clear, and less shotty pocks with little or no surrounding inflammation, and very rarely definite umbilication. They are numerous on the trunk, and do not as a rule involve the soles of the feet or the palmar aspect of hands or fingers. The stage of incubation is much shorter and much less marked.

Prognosis.—Almost invariably in recovery.

Treatment.—Relieve the itching in the same way as in small-pox. Prevent scratching, which may cause injury to the skin and permanent disfigurement. A soothing ointment, such as zinc oxide, and muffling up the patient's hands, if it is allowed, will usually obviate any trouble.

X DIPHTHERIA

An acute infectious disease characterised by the development of a fibrinous exudation most generally on the pharynx and in which the toxins produced by the organism cause marked constitutional symptoms.

Etiology.—The Klebs-Löffler bacillus is the specific organism present, although very frequently other organisms may be associated. The bacillus is non-motile, measures from 2-3 μ in length and about .5 μ in breadth, is very slightly curved, with rounded ends, and the ends sometimes stain in such a way as to suggest sporulation, which, however, is not present. Many variations in appearance are met with in the process of cultivation, but the bacilli almost always occur in the throat and elsewhere in irregular clumps, and they stain by Gram's, Neisser's, and other distinctive methods. Roux and Yersin separated the toxin, which was found to be as toxic to animals as inoculation with the organism itself.

The disease is transmissible by actual contact and probably not by the air, and therefore the use of common drinking-cups and kissing an infected patient would be at least certain, if not the most common, modes of infection. The disease however, has been gradually diminishing in frequency, owing to sanitary improvements, while the decrease in the death-rate depends to a great extent on the use of the antitoxin.

The specific organisms are found in the affected patches on the throat, and are sometimes discoverable on the fauces of perfectly healthy persons. Animals especially cats, appear to suffer from diphtheria. Milk is a frequent mode of infection. As already indicated, a mixed infection is not uncommon, and there is a distinct tendency for diphtheria to be associated with scarlet fever, measles, and whooping-cough, but it must be remembered that the appearance of a membrane does not always signify the presence of Löffler's bacillus. The disease is endemic in cities, epidemic at certain seasons of the year, and is most frequently met with between the ages of 2 and 15.

Pathological Anatomy.—The tonsils, soft palate, and uvula are usually affected, the membrane is at first adherent, and when scraped off leaves a raw bleeding surface (during

e). In later stages, however, and most frequently at the time of death, the membrane is readily separated.

The membrane consists of the superficial layers of the mucosa which have undergone coagulation necrosis, and in addition there are leucocytes and red blood corpuscles, fibrin, and many colonies of the specific organism. The diphtheritic membrane may involve the larynx and spread down to the bronchi, and even to the lung, producing septic pneumonia, while the lymphatic glands, which are always enlarged, may in some cases undergo suppuration.

In the *Alimentary System* there may be extensive involvement of the mouth, and even of the oesophagus and stomach.

In the *Hæmopoietic System* the spleen is usually enlarged and acutely congested.

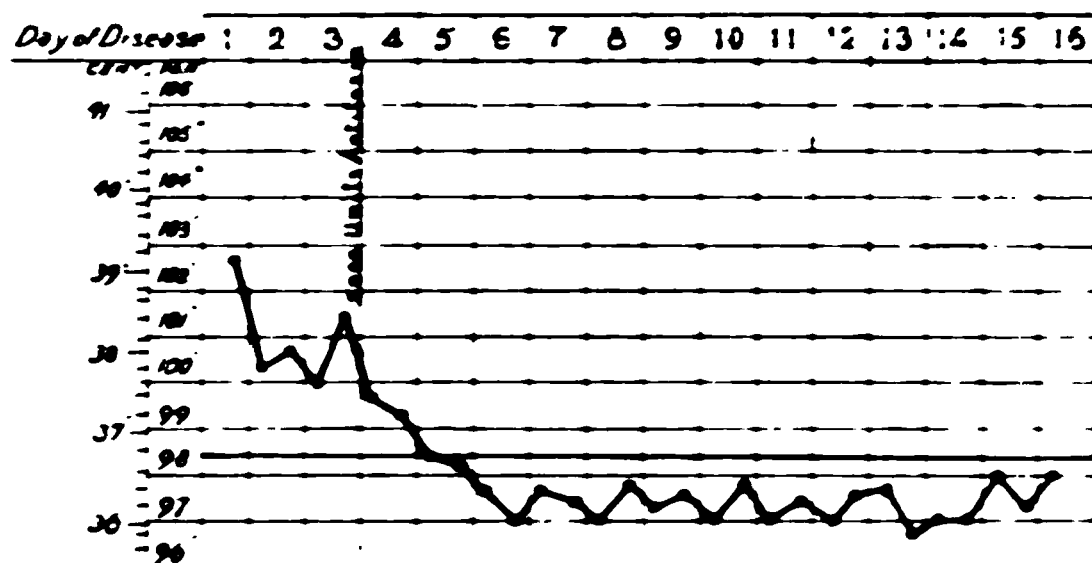


FIG. 10.—Temperature Chart. Diphtheria, showing effect of treatment on temperature.

In the *Circulatory System* an interstitial myocarditis has been described, while an atonic and fatty condition of the heart is a common result of the toxin.

In the *Respiratory System* septic pneumonia and pulmonary collapse may occur; while in the *Urinary System* nephritis is a somewhat rare complication.

In the *Nervous System*, in which paralytic phenomena result from the toxin, there may be the changes met with in ichthyomatous neuritis with consequent changes in the responding nerve-cells.

Hæmorrhages are not uncommon, just as in many malignant cases of fever.

Clinical Features.—Stage of *Incubation* 2 to 7 days. Stage of *Incubation* begins with marked constitutional disturbances, malaise, nausea, fever, etc., and the temperature

rises rapidly, reaching about 103° F. by the end of the second day, at which time the typical patch on the throat has generally formed.

Diphtheria patches appear on the tonsils, soft palate, and uvula, while in certain cases, without any pharyngeal affection, the larynx may be involved. The mucous membrane is first reddened and swollen, and greyish-white membranous patch or patches appear, covering a raw bleeding area, and the fauces become of a dusky-red colour. Often within a few hours the membrane begins to develop, although in certain cases it may be delayed, and it is important to remember that as the membrane is formed in part from the mucosa itself, it is at an early stage firmly adherent. The tongue is thickly furred, the glands of the neck become swollen and tender, and chewing and swallowing are extremely painful, and there is generally a trace of albumin in the urine. In a favourable case the membrane may cease to spread and gradually separate about the end of the 4th day, while in a more severe case it may persist till the 8th to the 10th days.

In all cases of diphtheria there is marked constitutional disturbance, but it varies in degree in different cases. Laryngeal diphtheria gives rise to attacks of dyspnoea, often paroxysmal and occurring at night. The distress may rapidly increase until the patient is asphyxiated, or the attack may temporarily yield. Much relief is obtained if the obstructing membrane is in part coughed up, when the croupy character of the breathing disappears.

The toxin exerts its influence in certain cases more than in others, and mainly on the heart and certain peripheral nerves, especially those which innervate the soft palate, the laryngeal muscles, more rarely the muscles of the eyeball, and still more rarely other muscles of the body.

Various types of diphtheria have been described, depending much on the position of the false membrane, whether in the pharynx, larynx, or nose, or whether involving a wound or the anus or vulva. A malignant type of diphtheria is generally due either to (1) a tendency to severe hæmorrhage from mucous membranes and elsewhere; or (2) an intense toxæmia, which kills the patient; or (3) extension of the membrane, as, for example, to the lung, producing a very grave diphtheritic broncho-pneumonia.

Deep ulceration and gangrene are also sometimes met with.

Complications.—In the *Circulatory System* heart failure is to be dreaded. It is probably due to the action of the toxin on the heart causing dilatation, but it may be the result of fatty degeneration, and a fatal syncope may follow even a slight effort, such as sitting up in bed, and rapid, irregular action of the heart should be considered a serious phenomenon.

In the *Alimentary and Respiratory Systems* we have only to note again the tendency to the spread of the false membrane down the alimentary and respiratory tracts respectively; while the frequent development of œdema of the glottis must not be forgotten in cases of laryngeal diphtheria.

In the *Urinary System* albuminuria is typical of the disease, and acute nephritis is not a very infrequent complication.

In the *Nervous System* the leading complication is paralysis, which reference has already been made. It is the result of the toxin, and occurs in 10 to 25 per cent of all cases of the disease. It usually appears 14 to 21 days after the attack, and may follow in the mildest case. The most common form is paralysis of the soft palate, causing a nasal type of voice and a tendency for fluids, which the patient drinks, to regurgitate from the nose. On inspection the soft palate is seen to hang absolutely motionless during both breathing and phonation, in addition it is insensitive to the touch. Less commonly there is paralysis of the muscles of the larynx, producing a tendency to asphyxia, because the openers of the glottis (the anterior crico-arytenoid muscles) are paralysed. Occasionally the eye muscles are attacked, causing squint, ptosis, and sometimes paralysis of the ciliary muscle; more rarely other cranial nerves are involved. The limbs are much less frequently affected, and only in a very few cases does paralysis of the costal muscles and diaphragm endanger life. The paralysis already indicated, a parenchymatous neuritis, generally gives a definite reaction of degeneration on testing the affected nerves with the galvanic current. There is a varying degree of anesthesia with, at first, a feeling of numbness and the pin-and-needles sensation. The paralysis usually disappears in 2 to 3 weeks.

Diagnosis.—In every suspicious case a swab from the

throat should be examined for the specific organisms. In the throat of *scarlet fever*, in *follicular tonsillitis*, and in *thrush*, the diphtheritic bacillus is not present, but it must not be forgotten that streptococci are often met with along with Löffler's bacillus. The membrane in thrush is whiter and more easily removed by scraping.

In diphtheria of the larynx the typical croup-like breathing, the albumin in the urine, and the obvious distress of the patient, render the diagnosis fairly easy even without a laryngoscopic examination.

Prognosis.—The death-rate is still high, and death is most apt to occur from heart failure, asphyxia, where the larynx is involved, or from extension of the membrane into the lungs; and in addition so-called malignant cases are nearly always fatal.

A large number of cases of diphtheria are now treated by the antitoxin method, and if this treatment is applied at a sufficiently early period, the chances of recovery are greatly increased, the death-rate rising rapidly each day which passes before the antitoxin is injected; in cases treated on the first or second day the death-rate is proportionately low.

Treatment.—The patient should be placed in a room with plenty of fresh air, and with the minimum of furniture; if possible, for cases of laryngeal diphtheria, a continuous spray of steam should be played upon the patient's face. The throat should be painted with the glycerinum acidi carbolici, diluted 1 part in 2 or 3 of glycerine, corrosive sublimate (1 in 1000 in water), chlorine water, or other antiseptic. In nasal cases the nostrils should be sprayed or washed out with antiseptics, and at an early period in most laryngeal cases tracheotomy or intubation must be performed, because asphyxia may rapidly develop and greatly endanger the success of the operation if delayed. Silver nitrate stick, or a 10 to 20 per cent solution in distilled water, applied to the affected part is of great value.

Quite as important as the local treatment is the careful feeding of the patient. Strong soups should be freely given, and alcohol, strychnine, and iron are frequently of great service. The risk of paralysis of the heart should ever be before the minds of doctor and nurse, and on no consideration should the patient be allowed to sit up in bed until all such risk has

away. Paralysis of the voluntary muscles (after a short of rest) should be treated by massage and a combination of galvanism and faradism. In rare cases, where there is paralysis of the respiratory muscles, artificial respiration has resorted to.

ANTITOXIN TREATMENT.—This consists in the injection of antitoxin serum of animals immunised against diphtheria. It is obtained from the horse, and the so-called "unit" is 100 times the amount of serum required to neutralise the minimum dose of the toxin injected into a guinea-pig weighing 250 gms. At one time 2000 to 3000 units were considered sufficient, but experience teaches that either this dose must be repeated more than once within 24 hours, or else a single dose of 1000 or 6000 units is administered, and a smaller quantity if necessary. The death-rate has been diminished by one-third as the result of this treatment.

Occasionally, following the administration of the antitoxin, urticarial or erythematous rashes appear on the skin, associated sometimes with fever and joint pains, and these may appear from 1 to 3 weeks after the injection. As a rule the antitoxin causes the extension of the membrane to cease, and the patient soon improves.

DIPHTHEROID INFLAMMATION

It has already been indicated that there are membranous inflammations involving the throat and larynx which are not caused by the Klebs-Löffler bacillus. Many of these result from the presence of the *Streptococcus pyogenes*, and the clinical picture may closely resemble true diphtheria. These cases are characterised by the formation of caseous deposits in the tonsillar crypts, or may be associated with one of the continued fevers, and especially with scarlet fever.

While many instances of so-called croup are in reality of hereditary nature, it is probable that cases in which a bacteriological examination has not been made, may be wrongly classified as diphtheritic, and such cases belong to the diphtheroid group.

The clinical features closely resemble diphtheria, and a local infection may prove quite as fatal in delicate children, although in the majority of cases so affected the cases are mild. It should be remembered that paralysis

may follow a streptococcal infection, just as it may result from true diphtheria, and the treatment carried out should be quite as thorough, and much on the same lines as that recommended for the more serious disease.

XI. WHOOPING-COUGH

(PERTUSSIS)

AN acute infectious fever, characterised by peculiar spasmodic attacks of coughing, terminating in the typical "whoop," and usually preceded by a period of bronchial catarrh.

Etiology.—Generally epidemic. It occurs almost exclusively in childhood, and before the age of 10; it is less common in tropical countries, and it is frequently associated with an epidemic of measles. Girls are more commonly affected than boys, and it is said that with them the death-rate is higher.

Afanassjev has discovered a short, thick bacillus, capable of producing the typical spasms in animals, and which he believes to be the specific organism of whooping-cough. The infection is certainly carried by the nasal and bronchial mucus, and therefore the clothes of the patient should be considered as infectious to other children.

Pathological Anatomy.—Bronchitis is always present, and there may be extension of inflammation to the lung, causing catarrhal pneumonia. From the strain of the coughing emphysema frequently results.

An attempt has been made to lay the blame of the typical spasm upon the swelling of the bronchial glands, or possibly the thymus; while it has also been suggested that the effect of the poison on the vagi nerves may explain the hyperæmia of the bronchi and the spasmodic coughing.

Clinical Features.—The stage of incubation is about 10 days. The fever may be divided into three stages:—

Firstly, a stage of ordinary bronchial catarrh commonly lasting 7 to 10 days, during which the usual clinical features of bronchitis become more and more marked, and there is a pretty definite coryza. The temperature is also as a rule slightly raised. Stress has been laid on a marked increase of lymphocytes appearing in the blood as an early sign of the disease.

Secondly, a stage when the typical spasm is superadded, remains present usually for a similar period of time. There are a great many short, spasmodic, expiratory coughs without any air entering the lung, the patient often becoming red in the face, followed by a long crowing inspiration, producing the typical "whoop" or "kink." A number of attacks follow each other, and generally relief is not obtained until either vomiting occurs or a considerable quantity of glairy mucus is expectorated. These paroxysms may be frequent, or occasional; they come on at night-time before sleeping, and after meals, almost constantly if the patient is excited, and are liable to be induced by hearing another patient cough. During the attacks many results of the great vascular reaction may occur, such as epistaxis, expectoration of blood, rupture of the tympanic membrane, convulsions, and emptying of the bladder and bowels. In an older patient there is a great danger of cerebral hæmorrhage. In most cases the attacks are so severe that the child rushes to the nurse or holds on to the bed, and in the infant the sufferer's face manifests great fear during the paroxysm.

Attention has been laid upon the presence of a small ulcer on the tongue, due to the lower teeth cutting the frænum during the spasm. These ulcers are very common, and occur in toothless infants.

Thirdly, during the next 10 days the spasm gradually subsides, and in a favourable case the child should be well again after a period of 3 to 4 weeks in all. In some patients there is a tendency for the spasm to persist. Should the child have a slight attack of bronchitis, an unusual typical whoop may reappear.

Complications.—The complication to be most dreaded is bacterial pneumonia, and for the prevention of this a great deal depends upon the capable nursing and treatment of the child.

Prognosis.—There is nothing which imitates the whooping-cough disease; even in *hysteria* the cough is rarely or never reproduced, but during the first stage of whooping-cough diagnosis is possible.

Prognosis.—The disease in itself is not serious, but when bacterial pneumonia follows (especially in young children) it is frequently fatal.

Treatment.—There are two methods of treating whooping-cough: (1) We can either, in perfectly strong children, treat them with fresh air, and in summer this is probably the most satisfactory plan. The child should merely be kept warm, properly fed, and otherwise be allowed to be as much in the open air as possible.

(2) In most cases, however, and certainly in cold weather, the patient should be kept exclusively in one room, or at least in one atmosphere. The air should be impregnated with a combination of creosote, guaiacol, or other soothing agent. Cresoline will be found admirably suited for this purpose, and the vaporising with this or similar drugs should be kept up night and day. Sulphurous acid fumes have been found useful, but they are most disagreeable to the attendants.

Internally much benefit is derived from giving fairly large doses of dilute hydrocyanic acid, bromides, chloral, and similar remedies. Bromoform in $\frac{1}{2}$ -1 minim doses dropped upon a lump of sugar has been specially recommended for controlling the paroxysms of coughing.

Externally gentle friction with camphorated oil, and sometimes the use of linseed poultices, are helpful, and in every case the chest should be covered with a Gangee tissue jacket. The child should be kept as free from excitement as possible, and the diet be such as to discourage the all too frequent tendency to vomiting.

XII. MUMPS

(CYNANCHE PAROTIDEA: EPIDEMIC PAROTITIS)

AN acute, infectious, febrile disease, characterised by inflammation of the salivary glands, especially the parotid; often occurring in epidemics, and sometimes, by metastasis, involving the testicles in boys, the breasts, and more rarely the ovaries, in girls.

Etiology.—A disease of childhood and adolescence, which is prevalent in spring and autumn, and is probably due to the specific streptococcus discovered by Micailis. It is highly contagious, and is said to be commoner in males. The patient is infectious for a period of about 3 weeks after the onset of the disease.

Pathological Anatomy.—The parotid and other salivary glands swell, the œdema being rather interstitial than parenchymatous in position. Rarely does suppuration result.

Clinical Features.—The stage of *Incubation* lasts from 1 to 3 weeks. The stage of *Invasion*, during which the fever is marked, the temperature rising to 103° or 104° F., accompanied by aching pains, especially in the salivary glands, and limited, as a rule, to one side of the face. The gland rapidly swells, not merely the part of the parotid in front of the ear being swollen, but the part also behind the ear of the ear. The gland has a somewhat elastic feeling on palpation, is tender to the touch, and is painful on moving the lower jaw, as in the act of eating. The pain shoots up to the ear, and sometimes tinnitus aurium is produced. One side alone may be involved, or the swelling may spread to the other side and there is usually marked salivation. The tonsils often enlarge, the breath is foetid, and the tongue heavily coated. The fever subsides about the 4th or 5th day, and the parotid swelling rapidly subsides.

Orchitis is generally the result of allowing the patient to get out of bed too soon. One or both testicles may swell and become extremely painful. The enlargement gradually subsides in a few days, and may be accompanied by a urethritic discharge. Orchitis is generally secondary to the parotid swelling, but may be primary, or even occur without any parotid swelling.

Metastitis occurs in girls much in the same way as orchitis in boys, but ovarian involvement is less common.

A considerable number of complications and sequelæ may arise, such as acute otitis, bronchitis, endocarditis, and so on, but with the exception of these complications due to stasis, the others are rare.

Diagnosis.—The condition is extremely typical, with the marked swelling in front of the ear on one side, while the fact of an epidemic being present should prevent any mistake.

Prognosis.—Recovery in a few days.

Treatment.—As a rule the patient should be confined to bed and especially if there be any temperature. Metastasis is generally the result of exposure to cold or wet, but it may be due to the patient's going about too freely even in the

The diet should be mostly fluid, as mastication is painful : attention must be paid to the bowels, and a fever mixture is often of value. Local fomentations with opium and chamomile flowers is an old-fashioned but useful remedy. Sometimes leeching has been resorted to, but it is rarely necessary. In convalescence the usual tonics should be administered.

XIII. RELAPSING, OR FAMINE, FEVER

A CONTAGIOUS epidemic fever in which there is no rash, but which is characterised by the presence of a specific organism in the blood, and by peculiar relapses of the fever after apyretic intervals.

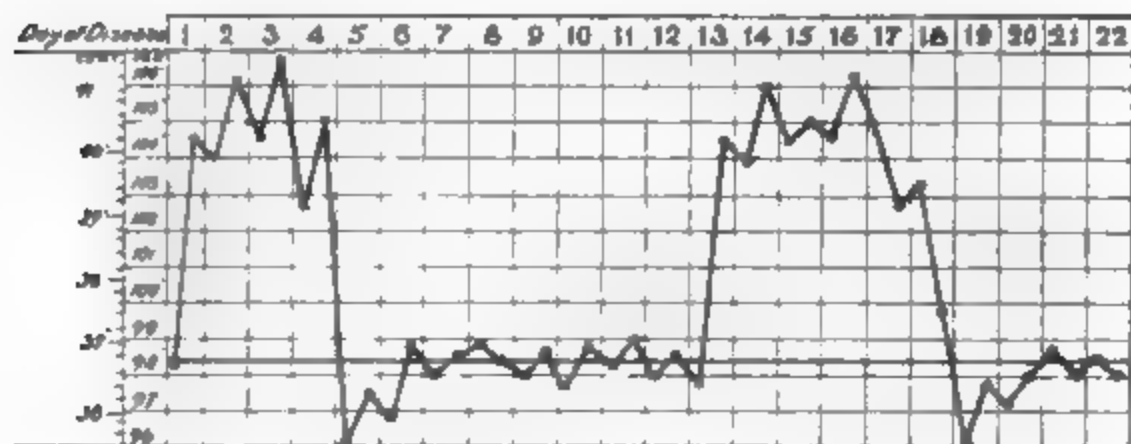


DIAGRAM 11 - Temperature Chart Relapsing Fever (After Murchison.)

Etiology.—Is caused by the spirillum of Obermeier, and is associated with starvation and overcrowding. The disease is infectious to persons well fed and well housed. The spirillum, which is about 2-6 times the diameter of a red blood corpuscle, is actively motile, and is found in the blood in great numbers. It stains well by Lottler's methylene blue stain, but has so far resisted all attempts at cultivation, although inoculation with the blood of a patient may produce the disease. The organisms appear in varying numbers from day to day during the attack, and disappear altogether in the apyretic intervals.

Pathological Anatomy.—The blood contains the spirillum, which multiplies rapidly, and the spleen is generally enlarged, but the disease is rarely fatal.

Clinical Features.—The stage of *Incubation* is 1 to 16 days.

The stage of *Invasion* commences suddenly, with rigors and shivers in the forehead, back, and legs. The temperature rises rapidly (103°-105° F.), and there is often vomiting, jaundice, enlargement, with tenderness, of liver and spleen. The face is red, the eyes congested, the tongue moist and thickly furred, and the skin is hot and burning. The pains increase, causing restlessness and frequently delirium. Suddenly on the 3rd or 4th or 10th days the stage of *Resolution* occurs by *crisis*, the temperature falling with profuse sweating. During the attack leucocytosis is usually present.

After a period of apyrexia, during which the patient may be perfectly well, in about a week's time a second attack occurs. Probably a third and even further relapses may occur, with apyretic intervals in between. Usually the successive attacks do not last so long as the first attack, varying in duration from 2 to 5 days. The crisis in each case closely resembles that occurring in the initial attack. Pains in the joints, with sometimes effusions, and in severe cases pulmonary and other complications, may be present.

Diagnosis.—The recognition of the spirillum in the blood is the surest test, but the typical relapses should prevent any possible mistake, especially as the fever occurs in epidemic form.

Prognosis.—Generally recovery, except in debilitated cases.

Treatment.—Common-sense rules should guide the physician in the treatment of his patient; sponging the skin, attention to the diet, and the use of an emetic where there is vomiting, are generally sufficient. Quinine has been tried as a means of preventing the relapses, but without avail. It should be administered during convalescence.

As a result of sanitary improvements, the disease is almost exterminated in our own country, and therefore little opportunity is now afforded of observing its clinical features.

XIV. SIMPLE CONTINUED FEVER

(FEBRICULA)

Simple continued fever, probably due to many different causes, is here described under this title. These cases of fever may be febrile, mild, and not pronounced attacks of one of the

ordinary eruptive fevers, such as scarlet fever or measles; or, *secondly*, they may be the result of poisoning from sewer gas, or possibly from the ingestion of some toxic article of diet. In addition to these well-recognised types there are, *thirdly*, cases of mild fever which follows a chill, generally disappearing in one to two days; and also, *fourthly*, cases of fever incidental to tropical countries, and perhaps due to exposure to the sun's rays. *Fifthly*, at the climacteric and in old persons mild attacks of fever without any definite cause are not uncommon.

The **Clinical Features** vary considerably, but rarely include pyrexia of more than 102° F., and the fever subsides in a few hours or days. A degree of malaise, headache, and loss of appetite are commonly present.

The **Diagnosis** is often important because of some infective fever being mistaken for this generally trivial ailment.

The **Prognosis** is favourable, and the **Treatment** consists in a simple diet and the usual febrile mixtures.

XV. INFLUENZA

(LA GRIPPE; EPIDEMIC CATARRHAL FEVER)

AN infectious disease, due to a specific organism, and specially affecting the respiratory passages.

Etiology.—Influenza has not infrequently followed attacks of plague during the later centuries. The bacillus was discovered by Pfeiffer in 1892, and it is present in the respiratory mucus. It is highly contagious, is probably conveyed by the atmosphere, and causes wide-spread epidemics. The bacillus is very minute, $.5\mu$ in length and $.2\mu$ in breadth. It is non-motile, grows well in broth, and is almost certainly the specific cause of influenza. While it spreads mostly by the air, it is quite probable that clothes and breath may communicate the disease to others. There is definite evidence that Pfeiffer's bacillus is not the only influenzal organism. It certainly is the cause of true influenza, but there are other varieties of the disease which are closely allied together as regards symptoms, but are dependent on different organisms.

Pathological Anatomy.—The post-mortem changes in

influenza are largely due to complications, such as pneumonia, to which the attack of Pfeiffer's bacillus appears to render the patient specially susceptible. The heart is apt to suffer, undergoing fatty changes or sometimes atonic dilatation, while there is a distinct association between the ravages of the tubercle bacillus and influenza. In what might be termed uncomplicated cases, a catarrhal inflammation of the respiratory passages and gastro-intestinal tract is probably the only fairly constant lesion.

Clinical Features.—The stage of *Incubation* is 3 to 4 days but possibly it may be a great deal shorter. The attack begins like an ordinary severe cold, with, however, more marked loss of taste and smell, often with bronchial catarrh

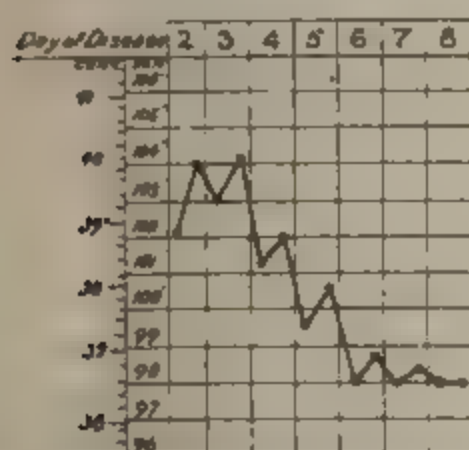


Diagram 12. Temperature Chart. Influenza. Recovery.

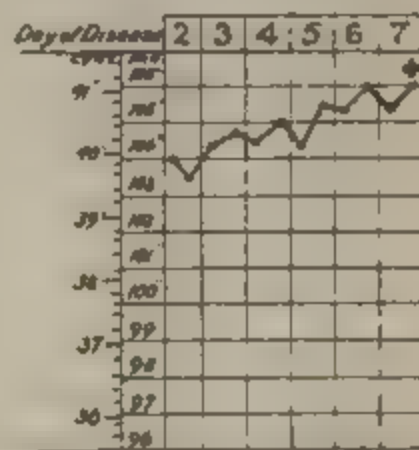


Diagram 13. Temperature Chart. Influenza. Death from cardio-pulmonary complications.

and sore throat, with headache, malaise, considerable pyrexia, pains in the back and legs, and very generally gastro-intestinal irritation. Certain patients suffer more in one way than another, some complain of photophobia, lachrymation, or ear-ache, while others suffer from cough and bronchial catarrh, and others again from nausea and sickness, with sometimes diarrhoea. It is common to find severe neuralgia either associated with the disease itself or as a troublesome sequel to it.

Various types of the disease have been described, such as the *Respiratory* type, the *Gastro-intestinal* type and types which indicate that the nervous system has specially suffered (*Nervous* type), or that the heart is seriously involved (*Cardiac* type).

Usually recovery occurs in a few days, but exposure to

cold or wet is very likely to induce one or more of the following complications:—

Complications.—Pfeiffer's bacillus predisposes to the attack of Fränkel's pneumococcus, and lobar pneumonia is an extremely common complication. In not a few cases the physician finds the patient's lungs attacked by a virulent form of pulmonary tuberculosis.

Cardiac failure, awful attacks of neuralgia, an excessive degree of prostration rendering all bodily or mental effort a serious tax, insanity, acute inflammation of the ear, and many other less common complications, may also be met with.

Diagnosis.—Unfortunately it is very difficult to find the bacillus of Pfeiffer, but the history of most cases gives a fairly sharp clinical picture. It should be remembered that there is no leucocytosis in influenza, and therefore *pneumonia*, which usually is associated with an increase of these cells in the blood, is, if of influenzal origin, accompanied by even a leucopenia.

In the tropics *dengue* is not unfamiliar, and has frequently been called tropical influenza. The back-ache of *small-pox* is certainly not unlike the pain of influenza, but the eventual appearance of the rash and the history of an epidemic in the neighbourhood should prevent any confusion. A common *cold* is not attended by the extreme prostration which marks influenzal patients.

Prognosis.—The death-rate from influenza itself is extremely small, only probably about 2 per cent, but it is proportionately more fatal in infants and in old persons, and the complications are very serious. Not infrequently the constitution of the patient is undermined, and it may take a long time before the effects of the fever are finally thrown off.

Treatment.—For the relief of the attack probably there is nothing so satisfactory as to give the patient, after a preliminary hot bath or hot foot-bath, a large dose, or several repeated smaller doses, of one or other of the following drugs:—Antipyrine, quinine, salicylate of soda, antifebrin, or phenacetin. These are the best of a long list of somewhat similar remedies, and they speedily relieve the aching pains and bring down the temperature. At the same time plenty of nourishing but light food should be given, and where there is any suspicion that the heart is affected, cardiac tonics must be promptly administered. Alcohol, and in large

es when it can be safely taken by the patient, is of enormous value once the pyretic period is past. Where, however, it is undesirable to order the patient alcohol—strychnine, nitrit of chloroform, and other stimulants and tonics should be judiciously exhibited.

The prostration and depression are often best alleviated by change of air, and, above all, by plenty of cheerful society.

PROPHYLAXIS.—During an influenzal epidemic many people are in the habit of taking ammoniated tincture of quinine in large doses, while eucalyptus and other volatile antiseptics may be smelt in the tramcar or the street. Certainly these remedies are advantageous, and relieve the anxiety of the persons who use them. A patient with influenza should be regarded upon as infectious, and the attendants and all delicate persons should endeavour by generous diet to keep themselves well above par when the disease is prevalent.

XVI. DENGUE

(BREAK-BONE, OR DANDY FEVER)

infectious fever, occurring in tropical and sub-tropical countries, associated with joint and muscular pains and typical eruptions.

Etiology.—The specific organism of this disease is as yet undiscovered, although the resemblance of dengue to influenza suggested a close relationship between the two. It occurs during the hot weather, it spreads with great rapidity, attacking a large proportion of the inhabitants of any district, and, like influenza, there is no protection against a second attack.

Pathological Anatomy.—The disease rarely endangers life, but joint effusions with pericarditis have been noted in some cases which proved fatal.

Clinical Features.—The *Incubation* stage is uncertain; the onset of *Invasion* begins with great suddenness, and generally shivering is followed by high fever, great pains in the joints and muscles, and severe headache, while the eyes are injected, the pulse is rapid, and a diffuse erythematous rash appears all over the body. The joints specially involved are

the knees, which become red and swollen, and the pains in the muscles and joints are increased by movement, thus giving rise to the term "dandy" fever.

In 1 to 3 days the fever subsides with sweating, but the pains in the joints persist somewhat, and after another period of 1 to 3 days' respite there is a rise of temperature and a second measles-like rash develops on the arms, legs, face, and trunk. After a few days it fades and desquamation ensues, sometimes accompanied by much itching and discomfort of the skin. There are a number of complications, and especially neuralgia and diarrhœa.

Diagnosis.—The history of an epidemic, and the absence of the profuse sweating which invariably accompanies *acute rheumatism*, should prevent any confusion with that disease; while in *scarlet fever* and *measles* the typical throat inflammation of the one, and the absence of joint pains in the other, should render a mistake improbable. In *influenza* eruptions are rare and catarrhal symptoms are much more common.

Prognosis.—The disease is rarely fatal, and recovery generally occurs in about a week to 10 days from the period of onset.

Treatment.—Sponge the skin and administer analgesic remedies, and especially phenacetin, while the patient should be kept in bed and given the usual fever diet. Baths, electricity, and massage do much for the relief of persistent joint pains, while iodide of potash has been frequently commended.

XVII. ERYSIPELAS

AN acute contagious inflammation of the skin, due to the *Streptococcus erysipelatis*, which gives rise to a varying amount of constitutional disturbance.

Etiology.—It is usually endemic, but epidemics are not uncommon at certain seasons of the year. It is a disease of cold or temperate climates, and is most prevalent during the spring and autumn months. The *Traumatic* type is met with in those who have pre-existing skin lesions, and is apt to occur in surgical wards. The *Idiopathic* form is probably the result of insanitary dwellings, but persons who are extremely debilitated, alcoholic, or who are suffering from Bright's disease,

ve a special predisposition. Certain individuals appear to ve a constitutional tendency to attacks. Infection is generally nveyed by direct contact.

Pathological Anatomy.—The specific organism closely embles the *Streptococcus pyogenes*, but it differs inasmuch it seldom produces suppuration. The affected skin is lamed, and specially so at the spreading margin where the eptococcus is present in large numbers. There is a large ount of inflammatory oedema with marked invasion by cocytes. Blebs are not uncommon, but suppuration is bably due to secondary infection by suppurative organisms. e spleen is enlarged and congested, and infarcts are found he lungs, spleen, and kidneys, where erysipelatous pyæmia supervened. In this case the pleuræ, pericardium, and

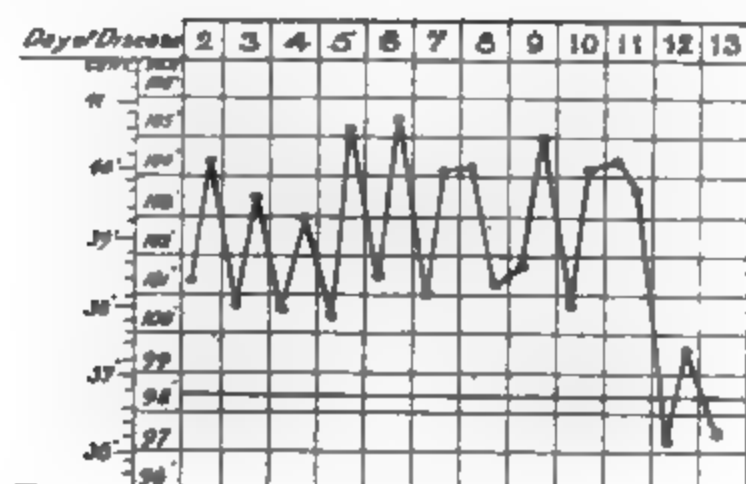


DIAGRAM 11.—Temperature Chart Erysipelas. Recovers

the endocardium may all be involved, and pneumonia acute nephritis are not infrequent. Where the scalp is ted, the meninges may become inflamed, probably through enous communications, and abscess formation is specially non in erysipelas in this region.

Clinical Features.—The stage of *Incubation* is 3 to 6

The stage of *Invasion* is accompanied by some malaise constitutional disturbance. During the stage of *Admanc* rruption the typical red, infiltrated area of skin appears,

on the face or round a pre-existing wound. The area on pressure, and has a distinct rampart-like edge, and

bullæ appear on the affected skin. The temperature to about 104° F., with all the usual febrile phenomena. lymphatic glands in the neighbourhood are often enlarged, there is a marked leucocytosis, and the temperature after

remaining fairly high for 3 to 5 or more days falls by *crisis*. When the face is affected, the features may be almost unrecognisable from swelling near the eyes. There is slight delirium at night, which is specially severe in bad cases, and in alcoholic patients albuminuria is often present. Where the mouth is the site of affection, the erysipelas may extend to the larynx, with sudden dyspnœa and death.

There is a special form of erysipelas called *Erysipelas migrans*, in which, as the name signifies, the rash spreads erratically over the body. It is a more chronic form, in which the temperature is very irregular.

Temporary alopecia is apt to follow erysipelas of the scalp. In certain persons repeated attacks occur, a pimple in the nose often being the exciting cause of a fresh outbreak.

Complications. — These have mostly been mentioned already. Suppuration, pyæmia, septicæmia, meningitis, acute nephritis, and various pulmonary and cardiac complications are not uncommon, and occasionally the involvement of the lymphatics may cause a condition resembling elephantiasis.

Diagnosis. — The rash is absolutely typical and characteristic of the disease.

The **Prognosis** is generally favourable, but likely to be fatal in debilitated or alcoholic subjects and in persons suffering from Bright's disease.

Treatment. — *Firstly*, endeavour to keep up the patient's strength by giving a light but nutritious diet, and alcohol is sometimes of great value. *Secondly*, apply to the affected skin either an ointment containing ichthyol (zinc-ichthyol salve - muslin, Unna), or else, where possible, subacetate of lead lotion. Carbolic and boracic fomentations are sometimes applied, and an attempt may be made to limit the spread of the inflammation by painting a ring of iodine round the affected area, or injecting various antiseptics into the spreading margin (corrosive sublimate gr. $\frac{1}{2}$). *Thirdly*, give internally full doses of tincture of the perchloride of iron (℥ 20-30), and where there is much delirium or excitement, order chloral (gr. 20-30) or opium (gr. $\frac{1}{2}$ -1).

In our experience anti-streptococcic serum administered by the rectum in 1 to 2 c.c., repeated every 2 or 3 hours, rapidly reduces the temperature, and appears to counteract both the local and general effects of the streptococcus.

XVIII. ACUTE PNEUMONIA

(CROUPOUS PNEUMONIA; LOBAR PNEUMONIA)

IS is a continued fever, due to certain specific micro-organisms, in which there is a characteristic inflammation of the lungs, and often a wide-spread infection of other organs.

Etiology.—The following organisms may be present:—

1. Fränkel's pneumococcus, or the *Micrococcus lanceolatus*, a diplococcus with a definite capsule, was recognised in 1884. It may be stained with carbol-fuchsin or gentian violet. There are certainly several varieties of this diplococcus which probably or possibly have different degrees of infectiveness and virulence. The organism is found in the sputum, in the affected lung, in the blood, and in the pleuritic exudates, where it is present in connection with the pericarditis, myocarditis, peritonitis, meningitis, and the synovitis associated with this specific fever. It has been found in the saliva of many individuals, but its relationship to lobar pneumonia is close and frequent.

2. Friedländer's pneumobacillus is much less commonly met, although it may be associated with the pneumococcus, and is more often found in broncho-pneumonias. It is also isolated, but shows totally different characteristics from the pneumococcus when cultivated.

3. The *Streptococcus pyogenes* and various staphylococci are also present in some cases, and mixed infections are not uncommon. We are not as yet in a position to state that the pneumococcus is the only specific organism of lobar pneumonia.

In addition to the organism or organisms present there are other etiological factors. Pneumonia is more frequent in autumn and winter, and seems to depend on exposure to cold and damp. Men naturally suffer more than women; and as regards age, from birth to the 10th year, and again from the 40th up to the end of life, pneumonia is of common occurrence.

There are certain predisposing conditions, and pneumonia usually follows influenza or other fevers, and attacks those who are debilitated and alcoholic. It may result from an injury to the chest, and it is apt to be the terminal

stage in diabetes mellitus, chronic Bright's disease, and various grave nervous diseases. *Very infectious.*

One attack predisposes to another.

Pathological Anatomy.—The distribution is generally lobar as regards the lungs, but a whole lung may be involved the affection often beginning at the base and extending upwards. It may be double, and is more often basal than apical. The right lung suffers more frequently than the left. Sometimes a lobular distribution occurs, but the lobar type is the usual one. Occasionally it begins in the centre of a lobe gradually extending towards the pleura.

Four stages are described:—(1) Congestion, which lasts probably 24-36 hours; towards its termination exudation begins. (2) Red hepatisation, lasting 2-4 days, in which the affected part of the lung becomes solid, and on section resembles red granite. The air vesicles contain a firm, fibrinous coagulum in which red blood corpuscles and degenerated endothelial cells are found. (3) Grey hepatisation, resembling grey granite, in which the lung becomes less vascular, and the coagula, which begin to separate, are invaded by numerous leucocytes. The lung is more friable, and some pathologists regard the term grey hepatisation as the stage in which breaking down of the lung is about to occur. In both red and grey hepatisation the pleura is involved, and a varying amount of exudate may be present. (4) Resolution. In a favourable case the lung clears up, mostly by absorption (digestion by enzymes), and partly by expectoration. This stage may be safely completed in 6-9 or 11 days. But in unfavourable cases the lung breaks down, and the cut surface on scraping exudes a thick muco-purulent material. Actual abscess formation may ensue, and even gangrene is possible.

In the affected lung the organisms are present in great numbers. The bronchial glands are generally enlarged, and these may soften and break down in fatal cases. In many cases after death extensive clotting is found to have occurred in the right heart and extending into the pulmonary arteries. This clot is very firm, generally adherent, and is characteristic of the disease. Purulent meningitis, pericarditis, endocarditis and peritonitis may all be present.

Clinical Features.—The Incubation stage varies, but probably about 1-2 days. Invasion begins suddenly, and

last 15 m to 1 hr

usually a rigor followed by a continued high temperature often accompanied by severe pain in the side. The patient lies on his back or on the affected side, with marked restlessness; the eyes are bright, the face anxious, and the patient works vigorously. There is marked dyspnoea, and the breathing is shallow. The

stitching pain
waite
abdominal

temperature is usually 103° F., and remains high, but it may swing gently with a slight rise at night. When resolution is reached, the temperature falls very suddenly, often within a few hours to normal. Occasionally the temperature falls gradually, or by lysis.

In some patients a pseudo-crisis occurs about the 6th

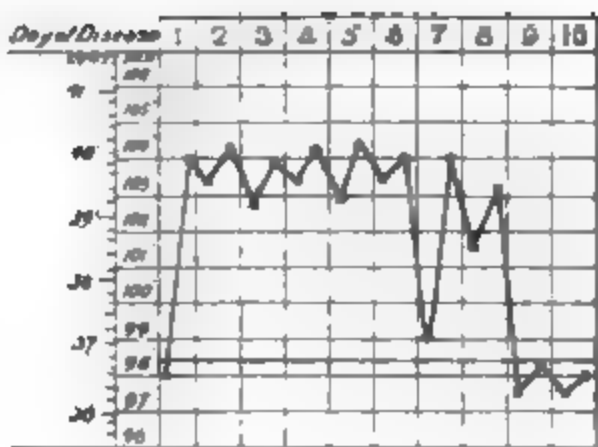


DIAGRAM 1. — Temperature Chart (Pneumonia), showing pseudo-crisis and later crisis, and eventual recovery

day, but the temperature rarely falls to normal, and it rises again to its former level within a few hours.

Respiratory System.—As a rule there is pain, and the respirations may rise to 40 or 60 per minute, the ratio to heart-rate often reaching 1 to 2. Cough is painful and ineffectual, although a little very viscid, rusty (blood-tinged) sputum is with difficulty brought up, and this increases in amount. The amount of blood varies and may be very considerable, suggesting a grave stage of the disease. The sputum contains an excess of leucocytes. It may become watery from oedema, and when serous character is assumed, the significance is grave. Bacteria are usually found in great numbers in the sputum.

Physical Signs.—During the stage of congestion the chest is not so dull, and the breathing is rather distant than of purely bronchial character. The physical signs of the consolidation stage are typical as a rule. Inspection shows the increased vocal fremitus is easily recognised. On percussion the note is dull over the consolidated area, but in the neighbourhood a tympanitic note from relaxed lung is to be made out. Auscultation yields typical tubular breathing, at first, very fine crepitations, later they may

Temp high
between 6
100-101
Side
2-3 m to 1 hr
100-101

2-3 m to 1 hr
100-101

Waller's sign

Waller's sign

Waller's sign

Waller's sign

Waller's sign

Waller's sign

Waller's sign

Waller's sign

disappear for a time, and then they become gradually coarser and bubbling (*crepitatio redux*). The crepitations have a sharp, clicking character. Very fine crepitations are limited to the first stage, and are thought to be due to the cohesion and subsequent separation of the alveolar walls. The vocal resonance is increased, and may be ægophonic in parts. All the auscultatory phenomena are temporarily modified by the blocking of the bronchus to the affected lung, should it occur. Pleuritic friction is often made out, and there may be evidence of a more or less extensive pleural effusion which may become an empyema.

Alimentary System.—The tongue is furred, often dry, and the edges red. Digestion is disturbed, and vomiting may occur. There is always malaise.

Circulatory System.—The pulse is full and bounding in strong patients during the earlier stages, but later it may become weak and dicrotic. Its rate is usually 100-120, depending much on the age and constitution of the patient. The first sound in the mitral area should be carefully ausculted; it early becomes less clear, and is replaced by a systolic murmur if the heart dilates, while sometimes irregularity develops. Special attention should be paid to the veins in the neck. Their distension suggests that coagulation of blood may be occurring in the right heart.

Hæmopoietic System.—The blood tends to clot, as above implied, because it contains an excess of fibrin, and there may be 10 parts instead of the normal 4 per 1000. There is usually a leucocytosis (mostly polymorphonuclear) reaching 20,000 to 40,000 per cubic millimetre of blood. If the leucocytosis is excessive, it is a bad sign, because it may mean breaking down of the lung; and if slight or absent, it indicates the inability of the individual to cope with the organisms present.

Integumentary System.—A burning skin is present during the fever, and copious sweating accompanies the crisis. Herpes labialis is extremely common about the 2nd day. It is said, but without much reason, that its appearance is a favourable sign.

Urinary System.—The urine is high coloured, with a dense precipitate of urates; the chlorides are diminished or absent. There may be slight albuminuria.

Nervous System.—In children headache and even con-

ons are common. Delirium is frequently noted, especially in alcoholic cases, and is very wild where hyperpyrexia

a. Meningitis is generally present in all cases, and may produce definite suggestive phenomena.

Locomotor System.—The joints are occasionally attacked by the pneumococcus.

There are many types of lobar pneumonia differing in a remarkable manner from the description given above.

There are Latent cases with little temperature; Migrating cases in which inflammation develops with extraordinary rapidity in one lobe, and as it disappears, while another lobe becomes involved. In Typical pneumonias there are often marked cerebral symptoms, and in Massive pneumonias the exudation is extensive, generally filling the bronchial tubes as well as the alveolar vesicles, while the greater part of a whole lung is involved. *Central Pneumonia* is also described.

Complications and Sequelæ.—It has been stated that an extensive pleural effusion, generally purulent, may ensue, and that changes may ensue in the affected part of the lung where resolution does not take place. The pneumonic abscess may form an abscess, or gangrene may develop, due to thrombosis of the branch of the pulmonary artery supplying that area. Fibroid changes may also follow, which do not clear up, and in certain instances the affected lung becomes infected with tubercle. Reference may also be made to ulcerative endocarditis, peritonitis, suppuration of the parotid gland, nephritis, and even meningitis, all due to the pneumococcus.

Diagnosis.—The definite physical signs characteristic of lobar pneumonia, consolidation with the discovery of the pneumococcus in the sputum are distinctive, but in the early stage when the consolidation is deep seated, the differential diagnosis between pneumonia, typhoid fever, acute tuberculosis, meningitis may be difficult. The Widal reaction in typhoid, the discovery of the tubercle bacillus, with the swinging or hectic temperature in cases of tuber-

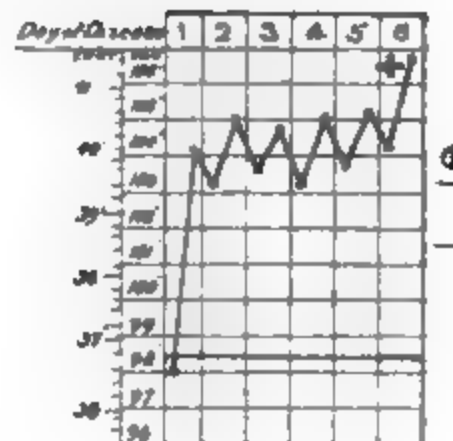


DIAGRAM 10. Temperature Chart. Lobar Pneumonia, with death from hyperpyrexia on sixth day.

culosis, are generally sufficient to clear up most cases. Meningitis early affects the cranial nerves, and where it is not simply a part of the pneumonic fever, a few days suffice as a rule to make the diagnosis definite.

Prognosis.—The death-rate varies (15-40 per cent) according to the virulence of the organism. The disease is more serious at the extremes of life, in alcoholic subjects, in delicate persons, and in those cases where the pneumonic attack is a final stage in some other serious disease, such as diabetes mellitus, Bright's disease, and cancer.

The following phenomena are of grave import in a case of croupous pneumonia:—a weakening heart with very rapid dirotic pulse, hyperpyrexia, a scanty leucocytosis, and probably also an excessive leucocytosis, a rapid spread of the consolidation in the same or to the other lung, and the development of edema or supervention of gangrene.

Treatment.—In the case of a strong patient no medicinal treatment may be required, and only the usual care with regard to food is needed, such as every severe infective fever necessitates, combined with attentive nursing.

Never let the patient sit up; insist on the prone position, and, if indicated, prescribe strophanthus or digitalis for the heart. Diffusible stimulants, and especially alcohol, are often given just at or after the crisis, and ammonia is believed to hinder the tendency to blood-clotting which is apt to be present in this disease. Locally much relief may be afforded by one of two methods of treatment: (1) In strong patients, where the fever runs high, apply ice or a cold-water coil (Leiter's tubes) over the affected lung. (2) For less robust individuals moist heat in the form of a linseed poultice, with or without mustard, is much more soothing, and the pleuritic pain, with the ineffective and harassing cough, is greatly relieved by this method of treatment. It is also well in such cases to use a steam bronchitis kettle to moisten the air which the patient breathes. Oxygen inhalations greatly aid the physician in combating the disease.

The usual cough, diuretic, and febrifuge remedies may be administered, but antipyretics should not be given freely, as they mask the true height of the fever. It is much wiser to control the temperature by sponging the patient, and, if there is hyperpyrexia, by wrapping him up in the sheet wrung out

Specific Infectious Diseases

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old or iced water, or using the cold-bath methods described on page 5, under the treatment of fevers generally.

Several curative sera have been recommended, but the success of the serum therapy of pneumonia is not yet assured. During recovery, or where resolution is delayed, counter-irritate with iodine, and remember not merely to give tonics, but to order pulmonary gymnastics.

*Mythology when used. Healer...
Simpler medicine - High Temp. of fever*

XIX. RHEUMATIC FEVER

(ACUTE RHEUMATISM)

Acute specific disease, characterised usually by joint swelling—sometimes by inflammation of fibrous tissue, and not infrequently accompanied by inflammation of the endocardium.

Etiology.—Acute rheumatism occurs most frequently during early adult life. More common in males than in females, it may occur at all ages and in both sexes. It is probable that its greater frequency in males is dependent on exposure to cold and wet associated with occupations.

It seems to be little doubt that a specific organism is the cause, and probably the diplococcus described by Poynton and Paine, and found in the blood and the synovial fluid of affected joints, is the pathogenic organism of rheumatic fever. Inoculation with this organism has produced endocarditis in animals with joint swelling. But inoculation with the specific organism is only one factor in the etiology. The predisposing factors of rheumatic fever are of great importance, and exposure to cold or wet, sudden changes of temperature, especially in connection with such employments as those of bakers, bricklayers, etc., may all act in this way. The disease is more frequent in damp climates, and is commoner in autumn whenever the weather becomes wet and cold.

Much discussion has arisen as to the hereditary element in the etiology. There is certainly much to be said in favour of a hereditary or nematic family history having, at all events, an important predisposing influence towards the disease.

A chemical theory for the production of the rheumatic fever has also been brought forward, and it was thought that

lactic acid might be the agent to which the pain and joint inflammation were due; but there is no evidence that any such chemical changes in metabolism have other than a subservient position.

Still a third theory, that the disease is one involving the trophic nerves, has little to commend it.

In conclusion, it seems probable that a specific organism is really present, and recent bacteriological research appears to support this view.

Pathological Anatomy.—There is congestion of the synovial membranes of the affected joints, with exudation of a synovial fluid, which may be clear or turbid, and which contains some leucocytes and an excess of fibrin. Only in the most

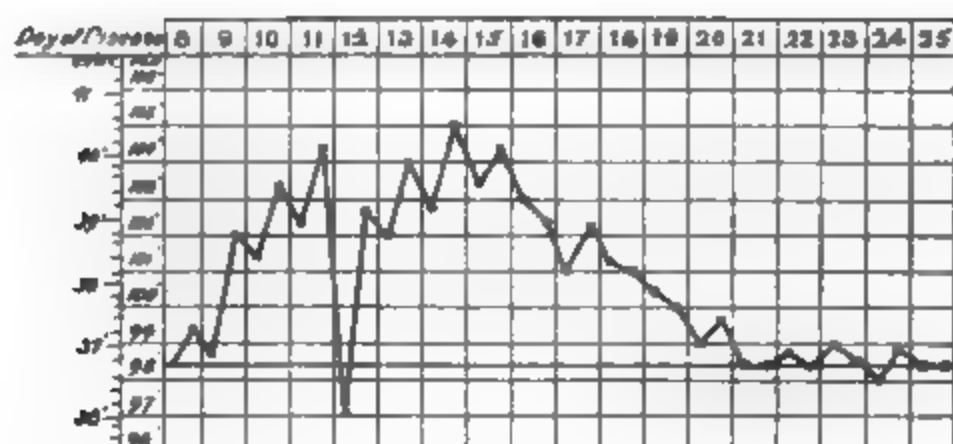


DIAGRAM 17.—Temperature Chart. Rheumatic Fever showing threatening of hyperpyrexia checked by cold bath. Recovery.

exceptional cases do the joints suppurate. The blood generally reveals a diminution in the number of red blood corpuscles, an increase of leucocytes, and an excessive amount of fibrin. The heart muscle is sometimes affected, probably as the result of the toxin, and this occurs apart altogether from the typical changes which are met with at the orifices in endocarditis. Pericarditis is also of frequent occurrence.

Clinical Features.—The patient begins to suffer from malaise, sometimes tonsillitis, and at an early period from pains especially affecting the joints of the arms and legs. The temperature rises to 102° or 103° F. or more, and usually there is a fairly definite morning fall. The pulse is bounding, the tongue moist and coated with white fur, while there is loss of appetite and great thirst. The joints most frequently affected are the knees, shoulders, ankles, wrists, hands, and elbows; more rarely the hips, toes, and other joints suffer.

joints are swollen, the skin being reddened, and the on, while mainly in the joint cavity, also affects neighbouring tendon sheaths and fibrous tissues. The pain soon becomes extremely severe, absolutely preventing the slightest movement, and in a most remarkable way it may change its location from one set of joints to another—joints previously affected being practically free from pain, and the swelling also spreading with great rapidity. Another typical feature is the profuse sweating which occurs all over the body. The peculiar sour odour, unquestionably due to the presence of lactic acid in the sweat, originated the idea that lactic acid might be the cause of the disease, but however profuse the perspiration may be, the acidity is due to accidental causes. Small clear vesicles from blockage of the orifices of the sweat-glands are not infrequently present over the trunk and especially the chest, and in certain instances some local irritations may arise round these sudamina.

Complications.—In the *Alimentary System* tonsillitis, as has been noted, is by no means uncommon, while gastric disturbance and constipation are generally present. But the *Circulatory System* demands the most careful attention. Endocarditis most frequently involves the mitral, sometimes the aortic orifice, and from day to day the heart sounds must be studied with the utmost care. Increase in the frequency of the heart's action, with perhaps a further rise of temperature, and very commonly palpitation, indicates in many cases that the endocardium is damaged: whilst the slightest change in the character of the heart sounds, even before a murmur develops, is important presumptive evidence in favour of commencing endocarditis. Pericarditis is also common, and the myocardium may suffer either from myocarditis, sometimes associated with endo- or pericarditis, or as a result of fatty degeneration. In the *Urinary System* there is an increase in the amount of urine and an excess of urates and urea common to any febrile condition; the chlorides are usually diminished.

There are certain complications other than those involving the heart, which are often intimately associated with rheumatic fever. In the *Respiratory System* pleurisy and bronchitis may be present; in the *Integumentary System* purpura, erythema, and eczema are by no means uncommon: while in

the *Nervous System* there is a close association between chorea and rheumatism, especially in children. We sometimes find that the rheumatic pains disappear and well-marked choreic movements take their place; and it has been suggested, although it is by no means proved, that endocarditis being common in both diseases may explain this association, probably by causing small emboli. Hyperpyrexia is a very serious complication not infrequently coincident with the appearance of pericarditis, the temperature rising to 107°-108° F. or more. It is accompanied by marked delirium and great restlessness, and, if it is not promptly checked, the patient soon passes into a stage of stupor ending in death. It has been called cerebral rheumatism, perhaps because with its appearance the joint pains markedly diminish.

Rheumatic nodules, which are small-shot or pea-like, subcutaneous, fibrous tumours, are found in the fascia or attached to tendons. They are certainly most common in children, and are met with on the fingers, hands and wrists, elbows, knees, spines of vertebræ, scapulæ, back of the head, and more rarely elsewhere. They are composed of round and spindle cells; as a rule they are not painful to the touch, and while also met with in the less acute types of rheumatism, they bear a definite, although not very explicable, relationship to endocarditis.

An attack of acute rheumatism usually subsides in about 10-20 days if untreated, but there is great risk of relapse, and endocarditis may develop during the primary attack or during a relapse.

Diagnosis.—It is almost impossible to mistake *gout* and *rheumatoid arthritis* for acute rheumatism, provided sufficient care is taken. Occasionally an acute rheumatoid arthritis simulates acute rheumatism, but the protracted nature of the disease soon clears up the initial uncertainty. There is a close resemblance, however, between *osteomyelitis* and *pyæmia* and this disease; but as a rule the bone inflammation in osteomyelitis is associated with a very marked leucocytosis, and to begin with the disease is more localised than acute rheumatism, while the migratory pains of acute rheumatism prevent confusion with the arthritis of pyæmia.

Prognosis.—Death is apt to be due to one of the complications so closely related to the disease, such as endocarditis,

arditis, pericarditis, or hyperpyrexia, while in uncomplicated cases recovery is usual. The risk of a relapse must not be forgotten, and the damage to the heart resulting from complications just mentioned may greatly interfere with life and usefulness of the patient.

Treatment. — The patient should be placed between blankets and clad in a flannel night-dress—these precautions being necessary to prevent chill from the excessive perspiration.

The night-dress should be slit down the front and down the sides, so that it can be easily changed, when damp, without disturbing the patient. The bed should be soft, but not a very soft mattress, the movements of which would greatly increase the agony of the patient. Absolute rest is essential, and the diet should consist almost exclusively of milk and occasionally soups, while plenty of alkaline mineral waters can be given to quench the thirst.

The proper medicinal treatment is much disputed.

1) We may give the salicyl compounds, generally administering the sodium salicylate in 20-grain doses 3 or 4 times a day and often in combination with sodium bicarbonate in similar doses. Salol (gr. 15-30) or salicin (gr. 20) may be used in place of the salicylate. With this treatment the pain is rapidly abated and endocarditis is possibly prevented, but the treatment is depressing, and the toxic action, especially of synthetically prepared salicylic acid, is distinctly dangerous. Cinchonism is not uncommon, and may cause great distress to the patient, and a trifling erythematous eruption sometimes appears in persons who have a peculiar idiosyncrasy to the treatment.

Much may be done to prevent these unpleasant results by variably using the salicylate prepared from vegetable sources, by clearing out the bowels before administering the drug, and by giving hydrobromic acid to counteract the tendency to cinchonism; but in cases in which the heart is markedly depressed it is wise to alter the treatment. Alkalies used to be administered for the relief of cinchonism in the belief that the disease was due to some poison, and much benefit is certainly obtained, as already stated, from the combination of an alkali with a salicyl compound. For children alkalies with salicin are most

usually the joints may be treated (1) by wrapping up in

cotton wool, or (2) by applying fomentations or poultices of a soothing nature. A useful application consists in smearing the affected joints with salicylate of methyl, combined with an equal part of vaseline and of lanoline, and covering over the joint with gutta-percha tissue in order to prevent the annoyance due to the penetrating odour of the drug. (3) Iodine may be painted on, either the ordinary tincture, the Edinburgh tincture, or equal parts of tincture and liniment. Counter-irritating liniments, or even blisters, may also be applied.

Nothing relieves the excessive pain of acute rheumatism so satisfactorily as a dose of Dover's powder, while phenacetin or kindred remedies may be cautiously administered.

Hyperpyrexia should be promptly treated; cold sponging, the application of the wet pack or the cold bath, must be resorted to without delay in order to save life.

In cases of endocarditis never give cardiac tonics unless when absolutely necessary: for serious affections of the heart or pericardium diffusible stimulants are more desirable, although they too should be given with caution.

Rheumatic fever invariably leaves the patient anæmic and prostrate, so that iron and general tonics, careful dieting, and the suitable clothing of the patient should all receive the attention of the physician.

Dr. Caton has recommended the application of small blisters along the course of the 3rd, 4th, 5th, and 6th dorsal nerves on the left side, in order to obviate or retard cardiac involvement, and the patient is kept in bed for a period of 6 weeks.

XX. EPIDEMIC CEREBRO-SPINAL MENINGITIS

AN acute febrile disease, characterised by nervous phenomena which indicate an inflammation of the cerebro-spinal meninges.

Etiology.—Considerable difference of opinion has arisen as to the specific organism responsible for this disease. Some authorities hold that it is the pneumococcus in the majority of cases, while the *Diplococcus intracellularis meningitidis* is held by others to be the probable specific agent. The disease occurs in epidemics and yet is not contagious, but children appear to be more susceptible than adults.

Pathological Anatomy.—An acute purulent leptomenitis of the brain and cord is present, with small hæmorrhages in the cortex, congestion of many organs, and sometimes adhesions into serous membranes.

Clinical Features.—There may be premonitory headache or malaise, but generally the sudden severe occipital headache, with rigors and pains in back and limbs, indicates the commencement of the fever. The head and neck are rigid and bent backward; there is great pain along the line of the spinal cord, cranial nerves, and often hyperæsthesia; convulsions, and finally coma and death, generally with a high temperature, terminate the scene. Delirium is a constant clinical feature, and may be of a wild type. Various skin eruptions, sometimes purpuric and sometimes herpetic, may appear, especially on the face, between the 2nd and 4th days. The purpuric spots are more marked on the trunk and limbs. There is occasionally optic neuritis, while there may be photophobia and sometimes paralysis of various cranial nerves. Cerebral vomiting is extremely common, and is an early symptom. A marked leucocytosis is present. There is often albuminuria. Various types have been described, depending on the duration of the fever or on its intermittent or other characteristics.

The **Sequelæ** are the results of interference with nerves and tracts, and include deafness, impaired vision, etc., and **Complications** such as pneumonia and pleurisy are not infrequent.

Diagnosis.—Probably lumbar puncture yields the most accurate information. The disease resembles *typhus*, especially regarding the rash, but the nervous symptoms are more marked in meningitis.

Prognosis.—May be rapidly fatal: epidemics vary in mortality from 30 to 70 per cent.

Treatment.—The patient should be isolated in a dark, well-ventilated room, but otherwise the treatment is the same as in an ordinary meningitis. The hair should be shaved and ice-bags applied to the head, the nape of the neck, and down the line of the spinal cord. Blisters are sometimes useful, and internal sedatives are of great value for relieving pain and excitement.

XXI. 1. SAPRÆMIA—2. SEPTICÆMIA—3. PYÆMIA

HOWEVER clear to the mind of the pathologist these terms may be, it is desirable that each should be carefully defined for the purposes of accuracy.

1. SAPRÆMIA

This implies the presence of toxins, but no organisms, in the general circulation, the organisms being entirely limited to some local site, as, for example, in tetanus and in many cases where there is a local focus of inflammation due to some suppurative or pathogenic organism. The poison may act with excessive power, not merely locally, but throughout the whole body, even although the manufacture of the poison is limited to a local site, and it is possible that an organism which may produce a sapræmia at one time, may cause a septicæmia or a pyæmia under conditions which must be different, although the exact nature of these differences is unknown.

The **Clinical Features** of sapræmia are chill and rigor, malaise, headache, thirst, sickness, and the result may be fatal if the poison be of a virulent nature.

2. SEPTICÆMIA

This implies the presence of organisms, as well as their toxins, in the blood-stream. There is thus a more wide-spread condition with many foci where toxins can be manufactured. The common organisms are the *Staphylococcus pyogenes albus* and *aureus*, the *Streptococcus pyogenes*, the *Pneumococcus*, and the *Bacillus pyocyaneus*. There may be a local wound, such as a post-mortem wound, with inflammation extending up the lymphatics of the arm, or there may be no definite site of any primary invasion. There are enlarged lymphatic glands in the one case; in the other there are no local phenomena to be noted.

Clinical Features.—There is greater malaise than in sapræmia, usually a higher temperature, which is sometimes hectic in character, a quicker pulse, and very generally marked

tro-intestinal disturbance. There is often a leucocytosis, spleen is enlarged, and we ought to obtain by culture the specific organism from the blood, although it may only be obtained with difficulty.

3. PYÆMIA

This implies not merely the presence of organisms in the blood, but large metastatic foci of organismal inflammation in distant parts of the body. There may be pyæmic emboli in lung, kidneys, spleen, brain, etc. The organisms may be the same as in septicæmia, and very frequently there is a local wound, a phlebitis, a sloughing uterine wall in the puerium, a carbuncle, an empyema, malignant endocarditis,

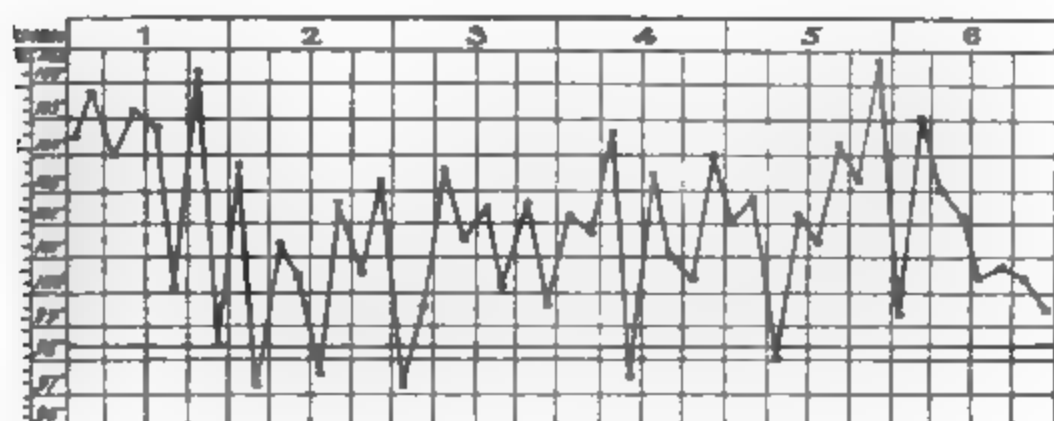


FIG. 15. —Temperature Chart. Pyæmia, showing the great variations of temperature.

osteomyelitis, middle-ear disease, or an ulcer in the intestine as the starting-point.

The organisms may spread by the lymphatics, by direct extension along bile-ducts, ureter, etc., or by infective emboli in the blood-stream.

Clinical Features.—Very similar to the last, but only more severe, and there is local pain where the septic emboli lodge, as for example in the spleen, kidneys, and lungs. The temperature is hectic, with marked rigors and sweating. There is malaise, there is always vomiting and diarrhoea, the appetite is lost, the tongue dry, and delirium precedes final prostration and collapse. Occasionally an erythematous rash is present, there should be a leucocytosis, and the spleen is enlarged. The evidence of pyæmic emboli is the following: albumin and blood in the urine, with sometimes local pain over the site, pain and perisplenitis in connection with the spleen

and the signs of broncho-pneumonia and pleurisy in the lung, while in cases where the endocardium is involved, fresh murmurs may develop.

It is rare to find the patient survive more than 7 to 10 days.

Diagnosis.—*Typhoid fever, acute miliary tuberculosis, and malarial fever* all somewhat resemble pyæmia; but Widal's reaction in typhoid, the examination of the blood, the benefit obtained from quinine in malaria, and the fact that in pyæmia a culture should be obtainable from the blood, ought to assist in clearing up the diagnosis. There will probably also be a leucocytosis in pyæmia (although this may not occur), and the spleen is usually enlarged. In all cases of pyæmia we should try to find the presumptive cause of the condition.

The **Prognosis** is essentially bad, and there is little hope of cure.

The **Treatment** of these three conditions—sapræmia, septicæmia, and pyæmia—is most unsatisfactory. In milder cases we endeavour to keep up the patient's strength, and quinine has been given with apparent benefit in some instances. It is certainly worth trying anti-streptococcic serum, which, if it does no good, will at all events do no harm.

TERMINAL SEPTICÆMIA

Closely allied to the conditions above described is the infection by organisms—sometimes local, sometimes general—found not infrequently in the terminal stages of certain diseases, and especially Bright's disease, heart disease, leucocythæmia, Hodgkin's disease, chronic tuberculosis, cirrhosis of the liver, and pneumonia. Thus the pleura, pericardium, peritoneum, and meninges may be affected in chronic Bright's disease, heart disease, etc., while the parotid gland sometimes suppurates in pneumonia, and an enteritis not infrequently is the cause of death in many of the conditions just mentioned. The attack of the organism occurs when the individual is much below par, and there is probably some special reason for the particular site selected, although the reason is by no means always clear. The organisms present differ considerably, but appear to be not infrequently the ordinary suppurative microbes.

XXII. CHOLERA ASIATICA

infectious epidemic fever, characterised by vomiting, purging, muscular cramps, and suppression of urine. It has an extremely high death-rate.

Etiology.—The comma bacillus of Koch was discovered in 1884. It is a minute, actively motile spirillum 1.5 to 2 in length by .5 μ in breadth, and several flagella of great length may sometimes be seen by special staining methods. The bacillus grows most actively between 30° and 40° C., and is killed by a temperature of 15° C. on the one hand and above 45° C. on the other. The organism produces in animals a disease similar to Asiatic cholera, and it is found in large numbers in the stools of affected persons. It is not communicated by the air, but is usually propagated by water or milk. The disease has been endemic in India for centuries; it has frequently spread to Europe, and this country has been visited by more than one severe epidemic. In the last great epidemic in Hamburg there were 18,000 cases (with 40 per cent mortality), due, it was believed, to the fact that the Elbe water, contaminated by sewage, was the main drinking-supply; while in a large suburb of Altona, where a better filtering system had been adopted, there were only 500 cases.

The disease has no respect for sex or age. It is certainly more common in tropical countries and during the warm season.

In many other fevers, drunkards present a much higher mortality rate, and there is a current belief that the disease may be induced to some extent by fear.

Pathological Anatomy.—Rigor mortis produces extraordinary effects after death. It comes on early, and often causes the limbs of the corpse to assume strange attitudes. A good deal depends on the stage at which death has occurred; but there is usually intense congestion of the intestinal tract, the contents being full of rice-water-like material, often unstained by blood, and Peyer's patches are swollen. Sometimes a diphtheritic membrane is present in the lower part of the small intestine. There is cloudy swelling in the kidneys, liver, etc., and during the stage of reaction in fatal cases there may be effusions of pleura, lungs, pericardium, kidneys, and other organs.

The bladder is usually empty.

Clinical Features.—The stage of *Incubation* is 2 to 5 days. The attack itself may be divided into three stages.

In the *First Stage*, diarrhœa, becoming more and more excessive, is the first symptom, while vomiting, colicky pain, and headache usually begin later; but there is rarely much fever. Towards the end of this stage the stools become rice-water-like in appearance, alkaline in reaction, have a specific gravity of 1006 to 1010, and contain sodium chloride, albumin, and mucus, and of course numerous organisms. Borborygmi are frequent, and epigastric pain, and intense cramps occur in the legs, especially in the calves, and eventually may involve all the muscles.

The *Second Stage* begins in 2 or 3 hours, the diarrhœa is much worse, and vomiting of the rice-water-like material commences. The pain is greater, the thirst more intense, and the patient becomes rapidly exhausted and collapsed, his skin being shrivelled and of a ghastly bluish-white colour, the eyes sunken, and every feature demonstrating intense distress. The surface temperature may fall as low as 92° Fahrenheit, while in the rectum it may be 103° or more. The pulse is almost impalpable, the first sound of the heart extremely feeble, and syncope frequently occurs during this stage, and is sometimes fatal. The urine is suppressed, hiccough may begin, the breath is described as being cold, and the patient is reduced to the most abject state of misery conceivable. This stage lasts from 2 to 24 hours.

The *Third Stage*, or stage of reaction, follows upon the preceding stage, should the patient still survive. The skin becomes warm, and is often covered with an erythematous rash, the pulse and heart improve, the diarrhœa and vomiting cease, at least for the time being, and the kidneys begin to secrete once more. The patient, however, runs considerable risk of inflammation of kidneys, lungs, and intestine, while the temperature may rise to a great height, and the patient die with marked nervous phenomena, such as delirium, subsultus tendinum, or possibly uræmia. Sometimes the diarrhœa continues, the stools becoming excessively fetid, or a relapse occurs, and if so it is very commonly fatal.

The complications have been already indicated.

Diagnosis. — *British cholera*, *dysentery*, or even *severe diarrhœa* where due to toxins, ptomaines, or arsenic, may be

rst readily mistaken for cholera; but a cholera epidemic in the neighbourhood, and the recognition of the typical miasm in the stools, usually clear up the case even in its early stage.

Prognosis.—The death-rate is 30 to 80 per cent. It is especially bad at the extremes of life, and debility and alcohol predispose patients both to the attack of the fever and also to a fatal termination.

Treatment.—The treatment of cholera resolves itself into two divisions: (1) prophylactic and (2) an attempt to relieve symptoms.

1) **PROPHYLAXIS.**—Patients should be removed to cholera hospitals, and the most thorough disinfection of clothes, drains, and cisterns should be insisted on, so as to stamp out the source from which the patient was infected. Remembering that the excreta are infective, they should be treated with carbolic acid or chloride of lime, and the condition of the water-supply of the community must be thoroughly investigated and any error promptly corrected.

A code of rules should be drawn up for the use of persons living in an infected district, and especially with regard to the use of water and milk, the cooking of vegetables and fruits, and careful attention to the prevention of diarrhoea. Every effort must be done to discourage alcoholic excess and to preserve the general health of the populace. Immunising experiments, as suggested by Haffkine, by injection with cultures of cholera bacillus have yielded good results.

2) **THE TREATMENT OF THE SYMPTOMS.**—The main thing is to check the diarrhoea, giving opium, bismuth, salol, and acetate of lead. Little benefit results from attempting to feed the patient during the stage of vomiting and diarrhoea, but iced effervescing drinks, and washing out the bowel and the stomach have been suggested. Severe cramps usually render this latter operation difficult and painful. Some benefit follows the intravenous injection of a saline solution (sodium chloride 1 drachm, and sodium carbonate $\frac{1}{2}$ drachm, to each quart of water), but it may be only temporary. In the stage of collapse apply heat by hot bottles and friction of the limbs, rubbing the patient with dry towels to remove the cold, clammy perspiration, and as vomiting ceases, give iced champagne, beef-tea, etc.

When the stage of reaction sets in, feed very cautiously, and try to keep down the temperature by sponging or similar means. General tonics and careful dieting are of much value during the stage of convalescence.

XXIII. SPRUE

A FEBRILE disease, characterised by irregular action of the bowels with frothy, pale, often pultaceous stools, flatulent dyspepsia, and a raw condition of the mucosa of mouth, tongue, and throat. It causes atrophic changes in the gastro-intestinal tract, with too frequently the eventual death of the patient, if not arrested by treatment.

Etiology.—This form of tropical diarrhoea is common in both the East and West Indies, and extends over a fairly wide range of country. It appears to be associated as a sequel with malaria, dysentery, and many affections of the alimentary tract. No specific organism has been isolated, but the disease, common to both sexes, is almost entirely limited to the foreign population of the lands in which it occurs.

Pathological Anatomy.—There is atrophy of the mucosa of the bowel, the glandular structures appear to suffer specially, while occasionally ulceration may be present, and the disease extends to the stomach and even the oesophagus.

Clinical Features.—A European acquiring the disease develops, in the first place, an ordinary diarrhoea, which soon becomes very excessive, the stools being profuse, pale, frothy, and often pultaceous, with much flatulent distension of the abdomen and a typical stomatitis. The diarrhoea once started progresses slowly, gradually undermining the patient's strength, and rendering the sufferer totally unable for work, physical or mental. The liver dulness is usually diminished.

A mild intermittent type of the disease is sometimes present.

Diagnosis.—The typical stools, together with the condition of the mouth and the country in which the disease was acquired, should assist in rendering the diagnosis easy.

The **Prognosis** depends partly on successful treatment, but mainly on the departure of the patient to a more temperate climate.

Treatment.—Rest in bed, with a rigid milk diet taken in small quantities, should be kept up for one to two months, and after, during convalescence, a somewhat more generous diet may be permitted. Opium, chalk mixture, and astringent diaries are valuable, but, above all, a change of climate greatly aids as a permanent cure.

XXIV. BUBONIC PLAGUE

CONTAGIOUS epidemic fever, generally confined to hot countries, with typical carbuncle-like swellings. It is probably the same as the Black Death of the 14th century, the name given to it on account of the hemorrhages with which it is associated.

History.—There have been outbreaks of plague in Europe in several centuries, and the great Plague of London in the 17th century was unquestionably a severe epidemic of this disease. It is associated with India, China, and the East, from some parts of which it is rarely entirely absent. Kitasato and Yersin discovered the specific bacillus during the Chinese epidemic of 1902.

It is a short rod with rounded ends, has a characteristic appearance in culture, and produces the disease when injected into animals. The bacilli are found in the blood,

lymphatic glands and viscera, and many animals, such as rats, and possibly even flies, may acquire and transmit the disease.

It is probable that the bacillus may live in the soil, becoming endemic in certain regions. It is not very contagious, and faulty hygiene is generally associated with most outbreaks. The organisms find entrance by the respiratory and digestive tracts, and possibly in some cases by inoculation. The attack appears to confer a certain amount of immunity.

Pathological Anatomy.—The lymphatic glands are inflamed, forming buboes. The spleen is enlarged and soft. Hemorrhages are common in connection with mucous and serous membranes. The post-mortem rigidity is often well marked, resembling in its effects the muscular contractions following an attack of cholera.

Clinical Features.—The stage of *Incubation* is 2 to 7 days. The stage of *Invasion* follows, in which prostration, headache and rigors are marked, and the temperature soon rises;

sometimes nausea and vomiting occur. The tongue becomes brown and dry, the urinary secretion is suppressed, and in two-thirds of the cases glandular swellings appear in 2 or 3 days in the groin, axilla, or neck. These swellings are extremely painful, and generally suppurate in about a week, while boils appear over other parts of the body. Sometimes petechial hæmorrhages come out on the skin in a severe case, and death may occur within the first week. The temperature during the later period of the fever may rise to 105° up to 107° F., and there are frequently extensive hæmorrhages from the nose, mouth, and elsewhere. The pulse, at first full, rapidly becomes feeble and dicrotic, and the spleen is usually enlarged.

The appearance of the glandular swellings frequently indicates that the stage of defervescence has commenced, and this occurs with profuse sweating. The glandular swellings discharge, and usually the central slough comes away. It is hardly necessary to add that *Resolution* is by *lysis*, and *Convalescence* may be retarded by prolonged suppuration and ulceration of the buboes, while complications and sequelæ, such as suppuration of the necrotic glands, pneumonia, and sometimes insanity, occur.

A number of clinical types of the fever have been described:—

(1) *A Malignant Type*, in which death may occur within 24 hours, with or without hæmorrhage, and in which no buboes develop.

(2) *Pestis Minor*—mild cases of plague, but with marked buboes, usually leading to a satisfactory result.

(3) *Pestis Major*—simply a severe type of bubonic plague.

There are also certain cases in which the lungs suffer specially, and to which the term *pneumonic plague* has been given.

Diagnosis. — The existence of an epidemic, with the characteristic buboes and the information gained by experimental inoculation of rats and other animals with the blood of the suspected patient, are usually sufficient to clear up any doubtful cases.

Prognosis.— The bubonic form is rather more favourable than the type in which no glandular swellings occur. Suppuration in the buboes is considered a good sign. The death-rate, however, is extremely high, from 40 to 80 per cent of cases proving fatal.

reatment.—The patient should be isolated, and strenuous measures made to secure greater sanitary cleanliness, and, perhaps, all, the absolute extermination of rats, to which the outbreak of the disease in a number of instances has been most closely traced. Not merely should the patients themselves be isolated, but their dwellings should be disinfected, and those who have been in contact with them quarantined.

As regards the patient, relieve the constipation, which is severe, by a dose of calomel, and thereafter give opium morphia, which will greatly aid in relieving the restlessness, delirium, and pain. Stimulants, and especially alcohol and quinine, are of great value where the heart is threatened to fail, and in no disease does iced champagne or some other tonic stimulant render such signal service. Above all, relieve the excessive pyrexia by cold sponging or other hydro-treatment.

The buboes should be treated, in the first place, by smearing with alcoholic extract of belladonna and glycerine in equal parts, and later they should be incised just as an ordinary abscess.

Many attempts have been made to introduce a satisfactory treatment, but without, at the present time, much success.

On the other hand, the immunisation of unaffected persons in a plague-stricken district is certainly worthy of consideration. Haffkine's serum consists of a sterilised bouillon and its efficacy as a prophylactic agent has of late been recognised. As already indicated, sanitary measures should be taken in a district in which plague has appeared, and every rat in the district should be destroyed. In addition, the general health of the populace merits the consideration of the authorities.

XXV. YELLOW FEVER

The specific tropical fever, characterised by jaundice, gastro-intestinal disturbance, hæmorrhages, albuminuria, and sometimes suppression of urine. Yellow fever is specially prevalent in the West Indies, in parts of South America, and tropical Africa.

etiology.—Sanarelli has isolated a specific organism,

which he describes as being a small ciliated bacillus from 2 to 3 μ in length, motile, and occurring in small numbers in the blood and tissues, and producing a very virulent toxin. The white population suffer more than the black, and strangers are peculiarly susceptible. Swampy, low-lying country, and especially insanitary and overcrowded towns suffer, and chiefly in hot weather. Apparently the organism possesses great resistance, and ordinary cold weather is not sufficient to exterminate it, the disease reappearing with any rise of temperature.

The mosquito is thought to be the medium of inoculation, and the disease cannot be carried by clothes or acquired by contact with infected persons.

Pathological Anatomy.—There is intense jaundice of the skin, and many petechial and other hæmorrhages are noted. There is marked fatty degeneration of the liver, often nephritis, and many red blood corpuscles are destroyed; otherwise the appearances are not distinctive. The supposed specific organism is found in the liver.

Clinical Features.—The stage of *Incubation* is 1 to 7 days or longer.

The attack is generally sudden, or after severe headache and malaise it begins with rigors. Great frontal headache, pains in the back and joints, and pain over the stomach, with nausea and vomiting, are prominent symptoms. In 2 or 3 days jaundice appears, and generally there is albumin in the urine. On the 4th day the fever remits, and the patient recovers, or a severe relapse may occur.

Especially in fatal cases, hæmorrhage from the stomach (commonly called "black vomit") and bowels is common, and there may be suppression of urine, convulsions, and coma. One attack appears to confer immunity. Recovery is often delayed owing to complications and sequelæ, such as protracted diarrhœa, inflammation of the parotid glands, nephritis, etc.

Diagnosis. — There is practically always an epidemic present in the district, but the appearance of the face, which is flushed and swollen, and the very definite jaundice, should help in the diagnosis. The fever most nearly resembling it is *blackwater fever*, but the previous history of malarial attacks renders a mistake unlikely.



Prognosis.—The death-rate is 15 to 85 per cent, generally near the higher figure. Much may be hoped from the action of a successful serum therapy.

Treatment.—The patient should be carefully treated, with the view of arresting the excessively painful engorgement, for which washing out the stomach, the application of blisters, and the administration of such remedies as hydrocyanic acid, creosote, or ice, are all recommended. Nutrient enemata should be given when vomiting prevents the use of food.

Hæmorrhage may need to be checked, and ergot, large doses of the tincture of the perchloride of iron, and acetate of iron may all be recommended. Small doses of $\frac{1}{60}$ th of a grain of chloride of mercury, administered in iced water every day have been found of advantage, and the free use of opium when necessary, helps to support the patient's strength.

PROPHYLAXIS of the disease is a disputed matter. Unquestionably the extermination of the mosquito may be hoped for; but sanitary improvements in crowded towns, the provision of a good supply of drinking water, and the prompt removal and disinfection of all discharges from the patient, are of great importance. At the present time it is difficult to say to what extent a rigid quarantine is a safeguard, but persons suffering from the disease should certainly be isolated.

XXVI. MALTA FEVER

(MEDITERRANEAN FEVER)

Malta fever, confined to places on the Mediterranean, and preceded by a long, somewhat tedious illness, with many relapses, the joints being specially involved, and joint pains very common.

Ætiology.—The disease is due to a definite organism, the *Bacillus melitensis*, discovered by Bruce in 1887. It is common in Malta, Gibraltar (Rock Fever), Naples, &c. It is not contagious, being probably transmitted from the animal nosphere, and originates from an insanitary drainage system. As might be supposed, it is most common in summer.

Pathological Anatomy.—The liver and spleen are enlarged, the latter being markedly congested; otherwise there is nothing to note.

Clinical Features.—The stage of *Incubation* is 6 to 10 days. There are various types of the fever which so far differ from each other.

(1) There is a type in which the temperature rises by a gradual daily increase, associated with marked pain, constipation, and headache. In about a week the fever abates and a period of apyrexia follows, which in turn is succeeded by another relapse and a small daily increase of temperature much like the original attack. These relapses recur sometimes for many weeks or months, and gradually they may become less marked and the patient recovers.

Associated with the disease are joint pains, bronchitis, and other complications.

(2) A malignant type may also be recognised, in which the fever process is very intense, and the patient dies from hyperpyrexia, cardiac failure, or possibly a pulmonary complication in a few days' time.

The **Complications** of the fever, some of which have been already indicated, are often very numerous and very severe: neuralgic pains, intractable insomnia, involvement of joints, orchitis, and many other less serious disturbances of the different systems, may render convalescence slow and unsatisfactory.

Diagnosis.—No fever presents such difficulty in regard to diagnosis, and it is chiefly by excluding everything else that one arrives at the belief that the case is Malta fever. *Typhoid*, *malaria*, and *rheumatic fever* are amongst the conditions with which the disease is most readily confused.

Prognosis.—The death-rate is low, being somewhere about 2 per cent; but, on the other hand, the long, protracted convalescence renders the fever more formidable than many conditions with a much higher mortality.

Treatment.—Dieting and attention to the patient's general comfort are usually sufficient. As soon as possible remove the patient to a region free from the disease, and endeavour to relieve the various complications as they arise. Quinine will be found of absolutely no use, and perhaps the

greatest relief, as far as the joint pains are concerned, is obtained from hydropathy.

XXVII. TROPICAL DYSENTERY

THE term dysentery is applied to cases of diarrhœa, with severe tenesmus, in which there is a typical mucous or mucopurulent stool, practically always at one time or other blood-stained.

While many cases in the tropics are truly amœbic, we frequently meet with dysentery due to the ingestion of unripe or over-ripe fruit, decomposing food, and other causes, and it is extremely difficult to dissociate entirely the two groups. In more temperate climates severe enteritis, if involving the large intestine, may acquire the characteristics of dysentery. To many of these sporadic and epidemic cases, not due to the amœba, the term bacillary dysentery has been given, because they are due to a definite group of bacilli.

Etiology.—Amœbic dysentery, by which is meant the specific tropical form of the disease, is almost certainly due to the *Amœba dysenteriae*, although other authorities claim that the presence of the amœba is merely accidental, and that some other organism or organisms may be the true cause of the condition. The amœba is a unicellular, protoplasmic, motile organism, 15 to 30 μ in diameter, with a clear outer zone called the ectosarc, and a granular inner zone called the endosarc. It contains a nucleus, and one or more vacuoles.

The bacillus of bacillary dysentery has been isolated by Shiga of Japan, and others. It is found in the stools, and is agglutinated by the blood-serum of the patient.

The disease is endemic in many lands, and specially in damp, low-lying countries near the equator; it occurs as an epidemic especially where large numbers of persons are collected together under somewhat insanitary circumstances, and is therefore frequently a scourge in military campaigns. The incidence of the disease is certainly lessened by careful drainage, attention to the water-supply, freedom from overcrowding, and care with regard to feeding of troops and others likely to be affected.

As already stated, unripe and over-ripe fruit and decomposing food either predispose to the specific form of dysentery or produce a severe form of enteritis indistinguishable from it.

Sporadic cases occur in both tropical and temperate climates, but where the specific organism, whatever it may be, suddenly attacks the individual, these cases are not so likely to prove truly infective to others. Probably the specific organism or organisms are introduced into the human subject in water, and in the autumn, or towards the end of the tropical rainy season, epidemics are most apt to occur. Both sexes suffer, no race is immune, and children appear to be specially predisposed. One attack of the disease renders the patient liable to future attacks, especially as the result of sleeping on damp ground, imprudent diet, or excessive fatigue.

Pathological Anatomy.—(1) In *Amœbic Dysentery* the large intestine is generally involved, with inflammation of the mucous membrane and specially the solitary glands, and, as a rule, the sigmoid flexure and upper part of the rectum are most markedly affected. Ulceration follows in the inflamed œdematous mucosa, and sloughs separate, leaving small, punched-out ulcers representing the solitary glands, and larger ulcers due to the ulceration of extensive areas of the mucosa. As a rule the ulcer does not involve more than the mucosa and submucosa, but the remaining wall of the bowel may be greatly thinned, and perforation through the muscular coat and peritoneum is by no means uncommon. Sometimes curious undermined bridges of mucosa join neighbouring ulcers together, and in some cases the whole mucosa becomes gangrenous, or may even be covered with a diphtheritic membrane. In these more serious types of the disease large areas of the mucosa may slough, and perforation or adhesion of the affected bowel to neighbouring structures may occur.

In the more chronic form of the disease there is a great amount of pigmentation, especially of the floor of the ulcers, and the affected areas are often greatly thickened, in other parts thinned, causing much alteration in the size of the lumen of the bowel, and stricture of the gut may result.

Secondary abscesses in the liver are specially frequent in amœbic dysentery, and may occur a considerable time after the onset of the attack; more rarely abscesses develop in other

3. In some of these abscesses the amœbæ may be found in the pus, which in the liver has a peculiar, brown-looking, often reddish appearance.

The mesenteric glands are inflamed, and may even suppurate, and in chronic cases are generally pigmented.

2) In *Bacillary Dysentery* there is intense congestion of the large intestine especially, but no ulceration as a rule. Sometimes, however, it occurs, the mucosa becoming of a blackish colour.

Clinical Features.—I. *Acute Catarrhal Dysentery, Amœbic and Bacillary.*—Malaise with a varying amount of fever, abdominal discomfort, and diarrhœa. The diarrhœa is frequent, with colicky pain and severe tenesmus. The tongue is at first coated, but later red; nausea and vomiting are occasionally present, and there is excessive thirst. The stools are typical, containing pus, blood, and glairy mucus; in the amœbic form the *Amœba coli* is present, and in the bacillary form large numbers of the *Bacillus dysenteriae*. During the active stage of amœbic dysentery masses of stinking, necrotic mucosa may be passed by the patient, and if gangrene ensue, the phenomena of acute obstruction are superadded in their worst form. These include intractable vomiting, the abdomen being swollen and excessively tender, and death terminates the attack in a few hours' time.

The number of stools varies greatly from 15 to 200 in the acute forms (according to the severity and type of the case), but in the amœbic cases relatively fewer stools are passed by the patient. The abdominal tenderness and the degree of inflammation present depend much on the severity of the process, and the amount of putrefactive change in the contents of the colon.

The temperature also varies greatly. In mild cases it does not rise higher than 101° or 102° F., while in more severe cases it may rise to 104°.

Occasionally a diphtheritic form occurs, with grave typhoid symptoms and high fever, which generally proves fatal.

Improvement may set in after a few days in mild cases, but in amœbic dysentery the disease continues for 6 weeks, eventually causing profound anaemia and debility. The bacillary form is very fatal when it is of a severe type.

Chronic Amœbic Dysentery.—The chronic form frequently succeeds the acute and has similar symptoms, although

less severe; there is less tenesmus, fewer stools, and the pain and discomfort are associated with the period following digestion when the waste matters have reached the large intestine. The appetite is capricious, and the patient may become anæmic and even cachectic from the long-standing drain on the system.

The bacillary form may also occur as a subacute or chronic disease, but less frequently.

Diagnosis.—There is no difficulty in recognising cases of dysentery, although it may be excessively hard to say whether it is the result of some dietetic indiscretion or a true case of the amoebic or bacillary type. The amoebæ and the bacilli should be searched for in the stools, and the fact that the bacilli are agglutinated by the patient's blood-serum should not be forgotten. Digital examination of the rectum should invariably be carried out where *hæmorrhoids*, *cancer*, or other local affection of the rectum is probable.

Prognosis.—The disease in its most favourable type is one of long duration, and it is extremely apt to recur, and therefore in all cases it is prudent to offer a guarded prognosis, although, if the sufferer leaves the country in which dysentery is common for a more temperate climate, the cure is likely to be rapid and permanent.

Treatment for both amoebic and bacillary dysentery.—Enjoin absolute rest in bed, with the most careful and restricted dietary, feeding the patient entirely on food-stuffs which leave, as far as possible, no waste matters to reach the large intestine. Probably milk and beef-tea in small quantities are amongst the best articles of diet.

The medicinal treatment is by no means satisfactory. We generally commence operations by giving a mild purge, such as castor oil, salts, or a small dose of calomel, and then attempt by one of three methods to relieve the patient. (1) Ipecacuanha powder in large doses of 20-40 or even 60 grains is given in wafer paper, and to prevent vomiting it may be preceded by a fairly large dose of morphia. It should be taken on an empty stomach and while the patient is absolutely at rest, because the slightest effort induces the rejection of the powder by vomiting. If the maximum dose has been administered at one time, 6 hours may be allowed to elapse before repeating the remedy. (2) *Sedatives* such

smuth, in quantities of at least 12-15 drachms in the 24 h, or *antiseptics* such as salol, in 15-20 grain doses, or chloride of mercury in $\frac{1}{100}$ grain doses every 2 hours, are of signal benefit. (3) An attempt may be made to kill the disease by rectal injections. After the administration of morphia or cocaine suppositories, irrigation of the bowels may be carried out with one of the following solutions: 1. 20-30 grains of silver nitrate to the pint of water, 2. pints being used, or antiseptic solutions of quinine, 3. 10 grains of quinine to 2000 of warm water, may be slowly injected into the bowels. It is hardly necessary to say that the silver nitrate injection is extremely painful, and the irrigation with quinine may cause the utmost agony to the patient. Some- times warm opium and starch enemata (60 minims of tincture of opium in 1-2 ounces of thin starch) are of great benefit in relieving the awful tenesmus, while washing out the bowels with warm boracic lotion often gives relief. Hot fomentations and hot fomentations applied over the abdomen are soothing.

In chronic cases, if at all possible, insist on the patient taking a holiday and returning for a considerable length of time to a more temperate climate.

XXVIII. BERI-BERI

Epidemic and epidemic peripheral neuritis, occurring in tropical and subtropical countries, but differing from the varieties of multiple neuritis by the special involvement of the vagus, phrenic, and vasomotor nerves, by great liability to cardiac dilatation, and by a tendency to cardiac dilatation which may

etiology.—The disease is found throughout a large part of the tropical world, but particularly in Japan, in the north of America, and in China, and it may also occur in temperate regions. It is certainly due to an organism, the nature of which has as yet been undiscovered. It is as common in the towns as in the country, and is certainly brought on by overcrowding, by heat, and by dirt, but appears to depend mainly on the diet of those affected. Persons living largely on rice run a great risk. Natives mostly suffer and not white people,

males are more affected than females, and the usual age is from 16 to 25.

Pathological Anatomy.—There is marked nerve degeneration closely resembling what is found in peripheral neuritis, and the vagus, phrenic, the cardiac nerves, and the nerves of the limbs and even the trunk, may be affected. In the muscles supplied by these nerves fatty changes are prominent, and the heart is greatly dilated, especially on the right side. There are, in addition, the usual changes in the nerve-cells of the degenerated neurones which one would expect to be present.

Dropsy is a distinctive feature, and may be present in most of the serous cavities, especially the pericardial sac, and also generally throughout the body.

Clinical Features.—The period of *Incubation* is unknown, but may extend for a long time. There are often premonitory symptoms, such as depression, numbness, headache, stiffness, and cramps, and during this period slight cedema of the ankles and feet, with a puffy appearance of the face, appears. Later a definite and pronounced peripheral neuritis develops, which rapidly becomes worse, the muscles are tender, while the skin may be numb, burning, or tingling, and cramps are not infrequent. The knee-jerks are lost, the affected muscles soon waste, and the patient becomes breathless and suffers from epigastric oppression and severe attacks of palpitation. The condition lasts for weeks or months, during which period the digestion may be unimpaired, the intellectual powers good, and there may be no fever of any kind. The affected muscles may show the reaction of degeneration until it is lost, and the patient frequently develops a typical ataxic gait with Romberg's phenomenon, but in any case, owing to the extent of muscular involvement, he becomes feeble and staggers when he walks. In raising the foot the heel is lifted first, and there is high-steppage gait. During all this time the cedema and general weakness become more and more marked, but cases vary in the degree of involvement of the heart, of the muscles, and even in the amount of dropsy present, so that many types of the disease have been differentiated. One remarkable feature is the absence of bedsores in nearly all varieties of this disease.

Death in fatal cases is generally due to the spread of the

ion in marked degree to the vagus nerve or the phrenics, cardiac failure, especially of the right side of the heart, to some dropsical effusion into the pleural or pericardial sacs, lastly, to prostration from the numerous relapses which occur.

Diagnosis.—As may be gathered from a careful study of preceding symptoms, the disease differs from ordinary *polio neuritis*, which is the only condition to which it bears obvious similarity, and of which it is in reality a special specific form.

Prognosis.—The death-rate varies from 1 in 40 to 1 in the patients attacked. Apparently cases with excessive rigidity are apt to be more serious, but epidemics vary greatly in mortality.

Treatment.—The food should be at once changed from that with which many authorities associate the disease, to a diet composed of meat, fish, fresh milk, and fresh vegetables, care should be taken to keep the patient from sleeping on damp ground, and to insure a dry, well-aired room. Drugs appear to be of any very great value, but quinine, salicylic acid, soda, arsenic, and many others have been recommended. Probably strychnine, administered by the mouth or hypodermically, together with electrical treatment and massage, will do more for the affected nerves and muscles than anything else.

Prophylaxis.—The prophylactic treatment is a question of the sanitary dwellings, and especially avoiding sleeping on damp ground, and most assuredly the alteration of the diet in rice-eating countries, where the disease is common, persons who live on a nitrogenous diet are exempt.

XXIX. TETANUS

(LOCKJAW)

The infective disease, characterised by tonic spasms of the voluntary muscles, and due to the presence of the bacillus of

History.—The bacillus was discovered over twenty years ago, and has been cultivated by Nicolaier and later by Kitasato.

It is an anaerobic bacillus growing in threads; the bacilli are sometimes motile and sometimes motionless. They are commonly known as the drumstick bacilli, because at one end of each bacillus there is a refractile spore, which rapidly develops and may be well seen in cultures of a certain age. The bacilli occur in the soil, the spores are very resistant, and as a result of the growth of the bacillus there is a virulent toxalbumin, which is said to be sixty times more poisonous than strychnine. Different observers have separated other toxic bodies.

Inoculation of a wound by the bacillus readily produces the disease, the phenomena of which are due to the production of the toxin or toxins. Probably infection is local, although the toxic bodies affect the whole system. The bacilli are found in garden soil, and are said to be associated in some way with the manure of horses.

A separate group of cases of the disease is called *idiopathic*, and to this group the cases reported as occurring in hot countries, and cases said to be due to cold, must be referred. An epidemic form called tetanus or trismus neonatorum occurs amongst newly-born children in hot countries.

Pathological Anatomy.—It is frequently stated that there is inflammation at the site of inoculation (if a scratch or wound), but it is difficult in certain cases to assure oneself of the truth of this.

In the central nervous system there is congestion, and the changes are most marked in the region of the great motor centres. It has been suggested that in the idiopathic form the spores may be inhaled, and that therefore bronchial irritation is present in those cases.

Clinical Features.—About 10 days after the injury slight stiffness of the neck and jaw muscles indicates the onset of the disease; the spasms increase, especially affecting the muscles of mastication, and the *risus sardonius*, or the typical and ghastly smile produced by the raised eyebrows and drawing outwards of the angles of the mouth, frequently develops. Gradually other muscles suffer, causing *opisthotonos*, during which the patient's body may be arched backwards, so that he rests on the back of his head and his heels. In other cases there may be lateral bending called *pleurothotonos*, or even bending forwards, to which the name of *emprosthotonos*

been applied. With the severe spasms the body is held in a vice, the pain is agonising, breathing becomes difficult, temperature rises often as high as 105° or 106° F., and death may occur from hyperpyrexia. In other cases death occurs during the spasms, or asphyxia, or sheer exhaustion, bringing about a fatal result. The slightest irritation, such as a heavy foot on the floor or shaking the bed, brings on the spasms. The pulse-rate rapidly increases, and there is the greatest difficulty in swallowing, and therefore in keeping up the patient's strength, while sleeplessness is a troublesome symptom.

LOCAL TETANUS is a term applied to the disease when it arises from a wound or injury in the realm of the 5th nerve. When the facial muscles become first affected, there is special difficulty in swallowing, and only eventually do the other muscles of the body suffer.

Prognosis.—*Strychnine poisoning* most closely resembles tetanus, but in it the jaw muscles do not suffer so early, and there is no rigidity between the spasms. *Tetany* is too slight to be a mistake possible.

Prognosis.—In milder cases, especially with the antitoxin treatment, recovery may occur, but in severe cases the death-rate is very high, and in so-called idiopathic cases the disease is usually fatal.

Treatment.—Attention must be at once paid to the condition of the wound, which should be excised and treated with antiseptics. Keep the patient in a dark room, absolutely at rest. Morphine, chloral, belladonna, cannabis indica, curara, and opium are all beneficial, while chloroform has been resorted to for the relief of the paroxysms.

Antitoxin treatment depends on the fact that Behring and Kitasato succeeded in rendering animals immune to the disease in a manner similar to what had previously been accomplished in the case of diphtheria. 20-30 c.c. of the antitoxin should be administered at once, and later 15-20 c.c. should be given. The method is still somewhat uncertain, but is well worth a trial, and especially so because the antitoxin has proved of much value in veterinary surgery.

XXX. HYDROPHOBIA

(RABIES)

AN acute disease depending on a specific virus inoculated by dogs, wolves, and rarely other animals, into the human subject.

Etiology.—The virus of rabies, although extensively used in the Pasteur method of treatment, is the toxin of an organism as yet undiscovered. It is a remarkable fact that whereas the virus is the same in all animals, and capable of causing hydrophobia, the bite of certain of these is more apt to be fatal than that of others. The wolf, fox, cat, and dog generally transmit hydrophobia, the wolf and the cat giving a more dangerous bite than the dog, the animal with which we most frequently associate the disease. The poison is in the saliva of the affected animal, and the certainty of inoculation depends greatly on whether the bite is on an exposed part of the skin, or whether much of the infective saliva is wiped off on the clothes. A lacerated wound is more apt to be followed by the disease.

Many other animals besides those mentioned may become inoculated, such as cattle and horses.

Pathological Anatomy.—Changes are described in the cerebral cortex, medulla, and spinal cord, and especially in the medulla, in the region of the vagus, hypoglossal and spinal accessory nuclei. The changes referred to are dilatation of small vessels, minute hæmorrhages, and aggregations of leucocytes, while the mucosa of pharynx and œsophagus may show marked congestion.

Clinical Features.—The stage of *Incubation* is of very varying length (weeks or months), depending apparently on the nature of the wound, the animal by which inoculation is produced, and the age and degree of immunity of the patient. The period is shorter in children, while certain persons appear to possess a remarkable resistance to the virus.

Long after the wound has healed the stage of *Invasion* manifests itself by renewed redness and irritation at the site of the scar, loss of appetite, and mental depression, which is soon replaced by excitement. There is irritability, especially

ght lights or loud sounds, and some stiffness of the throat es.

he next stage of the disease is one of marked excitement, restlessness and hyperæsthesia, with violent spasms provoked by any peripheral irritation, and especially affecting the es of the mouth, larynx, and pharynx; there is greatly in swallowing, because it produces spasm, and breathing difficult. The saliva cannot be swallowed, and is spat possibly in the faces of those standing around, and the ant bark with which the patient is popularly credited ly due to spasm. The temperature rises to 101° or F. This period usually lasts for 1½ to 3 days, and is led by the paralytic stage, terminating in 6-18 hours ie death of the patient, often by heart failure.

uring the greater part of the disease the mind is clear, gh hallucinations may be present in some cases.

agnosis.—The history of the bite many weeks or months together with the characteristic clinical features of the is sufficiently typical, but it is always desirable that posed mad dog should be kept alive and under observa- If, however, the animal has been killed, then a rabbit be inoculated under the dura mater with a small part dog's medulla. After a period of 2 or 3 weeks rabies s, should hydrophobia have been present.

agnosis.—The disease, once it commences, is generally and the success of the Pasteur method of treatment on the prevention of the attack rather than its cure. es of rabid wolves and cats are peculiarly fatal.

atment.—The wound must be at once cauterised and re applied above the part bitten; while sucking the if there is no abrasion of the mucous membrane of ith, should be energetically performed. As soon as the Pasteur method of immunisation should be carried

Pasteur's Method of Immunisation.—In 1885 Pasteur d a rabbit and inoculated under the dura mater a ount of the spinal cord of a rabid dog. In 15 days ppeared; one rabbit was inoculated from another incubation stage was reduced to 7 days. The spinal these rabbits were suspended in the air, and gradually : virulence. The cords kept for 15 days are first used

for inoculating the patient, and so on until a cord suspended for 1 day only is used, so as gradually to increase the immunity of the patient against the virus.

The risk is not a great one, but it is conceivable that an individual who has not really been bitten by a mad dog at all might acquire the disease by inoculation. The inoculation is generally performed into the abdominal wall, and is by no means free from pain, while subsequent pyrexia and malaise may be due to the treatment.

The death-rate from hydrophobia has been very considerably reduced, probably about 40 per cent of those actually bitten by mad dogs being successfully immunised.

When the attack has developed, little can be done, with the exception of keeping the patient at rest and administering morphia and other sedatives, while the feeding of the patient presents the greatest difficulty.

During the period of hot weather, when rabies is most common, dogs ought to be muzzled, and animals found about the streets, and which are unclaimed, should be destroyed.

PSEUDO-RABIES

It is important to remember that in hysterical patients hydrophobia may be simulated, and the better informed the patient is with regard to the clinical features of the disease, the more nearly are these imitated by the sufferer.

XXXI. GLANDERS

AN infectious disease occurring in horses, and due to the *Bacillus mallei*, causes glanders when affecting the nasal and respiratory passages, and farcy in the skin. Grooms are infected direct from the horse.

Pathology and Pathological Anatomy.—The bacilli are short and non-motile, and are somewhat difficult to recognise by staining methods. Typical swellings and nodules appear in the skin, mucous membrane, lymphatics, and also internally. These inflammatory nodules are composed of granulation tissue, in connection with which suppuration and ulcers result.

Clinical Features.—*Acute Glanders.*—After an incubation

of 3-4 days the temperature rises, and nodules, which ly ulcerate, are found in the nose, causing a copious ent discharge. The neighbouring lymphatic glands are ved, and death follows in 1-2 weeks.

Chronic Glanders.—Closely resembles a severe coryza; it e and difficult of diagnosis.

Acute Farcy.—Follows on inoculation of the skin, and sts in a localised inflammatory infiltration, producing taneous nodules along the line of the lymphatics (farcy , which rapidly suppurate. Neighbouring lymphatic glands oints are involved, and abscesses in the muscles follow, generally resulting from septicæmia in about 2 weeks.

Chronic Farcy.—Similar subcutaneous nodules, but with less inflammation, and suppuration is slower. The disease or months or years with varying results.

glanders not very infrequently a pustular eruption s on the face.

Diagnosis.—There is usually direct evidence of infection he horse, but it often runs a more chronic course in , and therefore may present some difficulties. In all a careful examination should be made of the secretion oculation experiments performed.

Prognosis.—In man it is a serious disease, only a small nt of really acute cases recovering, and even in the e forms at least 50 per cent die.

Treatment.—Prompt disinfection of the wound is abso- necessary, and an attempt may be made to apply tics to the nasal passages.

allein, a sterilised culture of the *Bacillus mallei* in peptone n, has been administered in minute doses to man, but it et impossible to say if it is beneficial.

XXXII. ANTHRAX

MALIGNANT PUSTULE. WOOLSORTERS' DISEASE)

ite infectious disease due to the inoculation with the bacillus.

round may be inoculated from infected cattle, as in the butchers, tanners, and woolsorters (malignant pustule),

while in the latter profession the bacilli may also be inhaled into the lungs, and there set up an internal and very virulent type of the disease.

Pathology and Pathological Anatomy.—The bacillus varies greatly in size, and is often as big as $20\ \mu$ in length by $1\ \mu$ or more in breadth. They form spores which are very resistant, and are probably the cause of infection of the respiratory passages or alimentary tract. Spores do not form within the body. The organism produces most powerful toxins, to which many of the clinical features are due.

In malignant pustule there is a rapidly extending area of inflammation round the site of inoculation. The central part of the pustule becomes necrotic, and the lymphatic glands in the neighbourhood swell markedly.

In the lungs it causes a type of broncho-pneumonia, and in any internal form of the disease it is not uncommon to find a wide-spread distribution of bacilli throughout the body.

The **Clinical Features** in the case of malignant pustule are a certain amount of pain, high temperature, and general malaise, while in the internal form the phenomena vary with the site of infection. In pulmonary anthrax (woolsorters' disease) there is local pain, severe cough, and at an early period in the illness great malaise and profound constitutional symptoms terminating in collapse. Where the alimentary tract is affected, vomiting and diarrhoea of an intractable type are superadded. Rapid death is inevitable.

Diagnosis.—In malignant pustule it is not as a rule difficult to get a fairly suggestive history, while a culture of the *Bacillus anthracis* should be made.

In the internal form the diagnosis is often extremely obscure.

Prognosis.—Prompt treatment of a malignant pustule yields the only satisfactory results. In internal cases there is little hope of cure.

Treatment.—In malignant pustule apply powerful antiseptics, such as liquefied carbolic acid, or a strong solution of chloride of zinc, or inject into the pustule a 10 per cent solution of iodoform in ether.

For the internal form support the patient's strength and prescribe quinine in large doses.

XXXIII. ACTINOMYCOSIS

Actinomyces or ray-fungus is common amongst cattle and it is rare in man. The exact mode of infection is doubtful.

The organism has given rise to much discussion both as to its nature and development. It is usually found in the affected tissue as an interlacing mass of radiating threads which at a later period develop somewhat larger club-shaped extremities. The fungus stains by Gram's method. In the areas involved by the fungus there is an extensive small-cell proliferation, and this occurs in non-resistant tissues, necrosis is apt to occur. In some places the disease spreads with great rapidity, but in others it is more or less limited, and pus formation is not always present, the pus containing the typical fungus.

The disease usually affects the tongue in animals, producing what is called "wooden tongue," and is most common in horses, but may be found in other domestic animals. In man it may involve the lower jaw, but in certain cases is limited to the head, often more marked in the lung, but may be met with throughout many of the internal organs, and it is difficult to say whether this extension of the disease is due to some focus of infection or whether it is primary.

Clinical Features vary greatly, depending on the extent of the infection. In the jaw the development of a suppurating abscess, which eventually discharges, is attended with a certain amount of pain, and possibly malaise, and it is probable that the constitutional disturbances are the result of the superadded attack by pyogenic bacteria.

The tongue is not infrequently affected, but it is certainly noteworthy that most instances of the disease in men are of the pulmonary form. The pulmonary form may resemble a bronchitis or a miliary tuberculosis, and sometimes simply a bronchitis. When the fungus is found in the sputum the diagnosis becomes clear. The liver and other organs are rarely involved.

Diagnosis.—Finding the ray-fungus in the pus is usually

sufficient, while the lymphatic glands suffer to a less extent in this disease than one would expect.

The **Prognosis** depends largely on the possibility of complete removal of the disease; otherwise it is eventually fatal.

Treatment. — Remove the disease by surgical means if possible. Internally the use of iodide of potassium (gr. 30-60 daily) has yielded in certain cases satisfactory results.

XXXIV. FOOT-AND-MOUTH DISEASE

AN acute infectious disease occurring in cattle, sheep, and pigs, and more rarely other animals.

Transmission to man may be by milk or butter from infected animals, or sometimes by inoculation, as by a scratch. It spreads in cattle with great rapidity, and usually after a short period of *Incubation* pyrexia sets in, and the mucosa of the mouth shows the typical inflammation.

The **Clinical Features** are inflammatory vesicles on the lips, mouth, and pharynx, which rapidly cause painful ulcers. There is fever, marked salivation, and generally considerable gastro-intestinal disturbance. In severe cases there is a tendency to hæmorrhage.

The disease usually terminates favourably, and the treatment should consist partly in the isolation of infected cattle, and as regards the human subject attention to general principles.

XXXV. LEPROSY

A CHRONIC infectious disease, caused by the presence of the *Bacillus lepræ*, and characterised by one or other of two definite types, either (1) a *tubercular* leprosy, in which tubercular-looking nodules appear in the skin and mucous membranes; or (2) an *anaesthetic* leprosy, in which the bacillus specially attacks the nerves, causing sensory, motor, and trophic changes. It not infrequently happens that a patient presenting the characteristics of the first type eventually develops the second, and *vice versa*.

Etiology.—The disease interests us specially because of its occurrence in India and many other of our dependencies,

in Europe, although less prevalent, it still exists in India and Sweden, and in parts of Russia, especially those bordering upon Asia Minor. In certain lands the disease appears to be much more infectious, as well as being more contagious.

Nansen originally discovered the bacillus of leprosy, but now its culture is extremely difficult, and the exact mode of infection in many cases is still mysterious. It can be cultivated, although this statement has been denied and reasserted many times. It can certainly be conveyed by contact, and therefore the segregation of lepers is an extremely important point in prophylactic treatment. Persons in contact with lepers frequently become infected, and evidence afforded by the heroic life of Father Damien, who devoted himself to lepers and lived in their midst, and who, after acquiring the disease, proves conclusively the contagiousness of leprosy; and this is no isolated instance. It is, however, possible for medical men and nurses to attend daily on lepers without becoming infected.

Spicy diet has been considered a possible means of preventing leprosy in those predisposed, or else it confers the protection towards the attack of the organism. The evidence, however, in favour of this theory is inconclusive.

Microscopical Anatomy.—The typical tubercular nodules consist of granulation tissue containing enormous numbers of the bacilli, partly intracellular and partly lying between the cells. These nodules involve the deeper parts of the skin and mucous membranes, and especially the conjunctiva, the cornea, and the larynx, and deep-seated ulceration is attended with the nodules. In the anæsthetic type of leprosy there is a form of neuritis due to the extensive infiltration of the affected nerves by the bacilli causing the trophic changes in the skin and other tissues.

Clinical Features.—*Type I. Tubercular Leprosy.*—There is a primary discoloration of areas of skin which are the seats of the tubercular nodules. These are brownish-red in colour, and are sometimes hyperæsthetic; they are characteristic of tubercular leprosy. In certain patients the pigment disappears and the skin becomes white, coincident with the appearance of local anæsthesia, while the hair over the affected parts falls out. At a later period the tubercular

nodules develop, and their distribution gives the face the peculiar leonine expression so characteristic of the disease. The forehead, just above the eyes, and the lips suffer specially. At the same time similar nodules appear on the mucous membranes of the mouth and larynx. Very deep-seated ulceration may follow; in one leper seen, more than half of the face was eaten away, the patient presenting the most ghastly spectacle imaginable. The eyes are frequently affected and sight is lost.

Type II. Anæsthetic Leprosy.—As a result of the attack by the bacilli on the nerves, pains, hyperæsthesia, and eventually numbness develop. Soon trophic changes ensue in the affected skin, and extraordinary contractions of fingers and toes may result, and owing to necrotic changes the phalanges may be gradually lost until the hand is completely mutilated. Bullæ form over affected joints preparatory to this destructive process. Macular spots, similar to those seen in the first type, appear on the skin of the body as well as on the limbs; but although they give rise to anæsthetic areas, tubercular nodules do not develop as a general rule. This type runs an extremely slow course, and many patients live for years, gradually becoming more crippled and unable to use their hands and feet.

A combination of the two forms, as already indicated, is by no means rare.

Diagnosis.—The macular eruption is very typical, and especially when the anæsthetic stage has been reached. In the tubercular form of the disease the leonine appearance of the patient is almost distinctive. It is impossible, with care, to confuse the perforating ulcer of the foot of an *ataxic* patient with leprosy.

Prognosis.—In the anæsthetic type long life and even comparative usefulness is assured, while there is a feeling that this type is less infectious to others than the tubercular form, in which ulceration is so much more constantly present.

Treatment.—There is no remedy which can be truly called a specific for this disease. For many years certain of the essential oils peculiar to India and other lands have been recommended. These include gurjun and chaulmoogra oils, used both internally and externally. Much was hoped from tuberculin, which seemed to give favourable results in one or

ases, but it is now proved to be of no real efficacy. enene (Calmette) is at present being tried, but it seems ole that successful treatment must depend on serum y. Careful segregation and the prevention of marriage n lepers and healthy persons have done much to stamp orsy in Western Europe, although the problem remains ous as ever in India and many other countries.

XXXVI. SYPHILIS

ASE due to a specific organism or organisms, the exact of which is still doubtful. It may be inherited or d by infection, and both the ovum and spermatozoa able, the first of being infected from the mother, and ond of carrying infection. In syphilis there are three

The *primary* stage is only found in acquired cases, e manifestation is the appearance of a local primary the site of infection. The *secondary* stage follows in eks to two months' time, when rashes appear on the id superficial sores on the mucous membranes. The *tertiary* stage succeeds the secondary at an interval of nths to several years, and in it deep-seated granu- s tumours develop in the muscles and in many parts ody.

hould be stated definitely that syphilis is quite distinct lthough it may be associated with, soft sores (soft s) and gonorrhoea.

ology.—Lustgarten, as long ago as 1884, described a which is acid-fast like the tubercle bacillus; by this t that if stained with, for example, carbol-fuchsin, it he action of the acid decolorising agent, whether nitric uric acid solution is used. There is an even closer nce between Lustgarten's bacillus and the smegma a fact which is suggestive of the source of error, if the y is proved incorrect. More recently other organisms en isolated, and especially Schaudinn has described a te actively motile, very difficult to stain, and g in great numbers in the discharges and the tissues

modes of infection are certain, although very various.

The disease is most often acquired during sexual intercourse, probably due in some cases to a slight local abrasion, in others to infection through healthy mucous membrane; but kissing, sucking the nipple, vaginal examination where there is a lesion of skin on one of the examining fingers, circumcision where the operator is syphilitic and performs the rite with the teeth, and vaccination from a syphilitic child, are all fairly common methods of communicating the disease. Dirty dental instruments, drinking-cups, smoking the same pipe as a syphilitic patient, are also a few of the other ways by which infection may be conveyed. The primary sore, the condylomata, and the mucous plaques are intensely infective. Blood is probably capable of conveying infection, but saliva, tears, sweat, and milk are in themselves harmless unless mixed with discharge or blood. The semen is said to be innocuous, but this is very questionable, as the spermatozoa can almost certainly transmit the disease. In hereditary syphilis the discharges from syphilitic sores are infective. It is stated that the secretions from tertiary sores are non-infective, but this is more than doubtful, although they are certainly not so virulent. In inherited syphilis either the father or mother may give the infection, and a syphilitic child may be born by a woman who is not herself affected, but in such a case, according to Colles' law, the mother is protected against syphilis, although the child in suckling may give the infection to a wet nurse. Similarly, a healthy child born of syphilitic parents is said to be immune, but this statement, made by Profeta, requires more circumstantial evidence to prove its truth. Syphilis acquired from the mother is always more severe than infection from the father.

There are some curious facts about syphilis and immunity. Certain peoples and races appear to be becoming saturated with the disease, so that in them it runs a less severe course. In other races not previously exposed to it, syphilis, if introduced, assumes a most malignant type, and where it is transmitted from an individual of one race to a member of an entirely different race, it is apt to appear in an aggravated form. Should an individual have one attack of syphilis, he is usually protected against a second attack, and, if reinfected, the manifestations are generally much milder; rarely is the converse of this true.

Pathological Anatomy.—I. The *Primary Sore*, or hard chancre, consists of a great infiltration of the deeper parts of the skin with small round cells. There is proliferation of connective-tissue cells, and the small blood-vessels undergo endarteritis obliterans. The cell proliferation appears greatest in and around these small vessels, and there is an indurated edge due to cell proliferation and exudation. Old chancres tend to break down, and, as already noted, the edge is very infective. The glands in the neighbourhood become inflamed and become indurated. In old chancres there may be seen the epithelioid cells and even the giant cells so commonly associated with tubercle nodules, but they are not seen in syphilis.

The *Secondary Affections* are very various. There are superficial ulcers on the tonsils and pharynx; these are often circular, and whitish patches appear on the tongue and buccal mucosa, and are most marked in smokers. These lesions denote small-cell infiltrations, and they are due to the syphilitic virus in the blood. The secondary roseolar rashes are due to hyperæmia, and infiltration with small round cells. Spirochaetes-forming organisms may be present in addition, leading to pustular eruptions more or less local. Iritis is a common secondary lesion. These secondary lesions are less infective than the primary chancre, although they may transmit the disease, and a patient is not considered to be cured of syphilis until at least two years have elapsed since the last manifestation disappeared.

The *Tertiary Affections* are rarely seen now, owing to the prompt and vigorous treatment at earlier stages.

The essential lesions are gummata or tumours, made up of proliferated connective tissue, which may be distributed in a most irregular fashion throughout the body. They may occur anywhere, and may be soft or hard, minute, or as large as a small tumour.

The centre tends to become gelatinous-looking, or caseate; the periphery is firm and fibrous. This is due to central degenerative changes and thickening of the fibrous capsule is typical of older gummata, and causes remarkably deep cicatrices seen, for example, in the liver. Any two or three of these may occur together. They are common in the skin, bones, periosteum, and internal organs, including the meninges of the brain. Superficial gummata are apt to

break down and form ulcers; the internal ones remain long quiescent, and eventually may be absorbed, leaving a puckering or scar, or may become the site of calcareous deposition. Gummata tend to appear wherever the circulation of blood is least active. They are met with in the septa of muscles, and in fibrous structures in connection with joints. They may be found in the fibrous perichondrium of the larynx and in the median septum of the tongue. They also occur where there has been an injury; hence the syphilitic nodes on the tibia, and the development of gummata about a joint which has been bruised or overstrained.

In the skin tertiary lesions are deep seated, and lead to permanent disfigurement from cicatrisation.

In connection with the arteries there is endarteritis obliterans, in which the inner coat undergoes enormous thickening, usually at one side. The newly formed tissue of spindle-shaped cells develops just internal to the endothelial cells of the intima, and only in certain cases is there a marked cellular invasion of the other coats of the vessel. The arteries most generally affected are the cerebral vessels, and the disease may go on until thrombosis occurs from interference with the lumen. Endarteritis obliterans develops sometimes in hereditary syphilis, but generally in the acquired form and in the tertiary stage. In some cases a syphilitic periarteritis is present, in which there is gunmatous formation, especially in the adventitia.

Amyloid or waxy degeneration is a condition commonly associated with tertiary syphilis, and about one-third of all cases of waxy degeneration are of syphilitic origin.

Lastly, there are many distinct but late results of syphilitic toxins which may be properly referred to here. These specially involve the nervous system, and, as probable instances, locomotor ataxia, general paralysis of the insane, and epilepsy in some of its varieties may be cited. Although doubt exists as to this relationship, there seem to be well-grounded reasons for associating not a few cases of these and kindred nervous diseases with syphilis.

Clinical Features of Hereditary Syphilis.—The child at birth may be hall-marked with the disease. The chief phenomena are snuffles, fissures at the angles of the mouth, or sometimes at the anus, and very generally an eruption on the

In most cases, however, the child is merely puny, and characteristic features only develop about the 4th to the 6th weeks. In other instances the child is born healthy, but does not grow well.

1) Snuffles is always present, and is amongst the first most obvious symptoms. It may be so severe as to interfere with breathing when the child is at the breast, and the discharge may become purulent, and eventually the cartilages and bones necrose, which gives the peculiar flattening of the root of the nose so characteristic of congenital syphilis. Sometimes the Eustachian tubes are involved, and deafness results.

Skin eruptions are seen. These include the typical coppery rash at the angles of the mouth and mucous patches on the inside of cheek, pharynx, and tongue. The secretion from these is virulent, and may easily infect a wet-nurse or another individual, although not the mother (Colles' law, *vide* supra).

There are in addition typical copper-coloured, erythematous rashes, well seen about the nates, and the hair may

The permanent upper incisor teeth show the peculiarity described by Hutchinson. They are peg-shaped, short, and have a notch or groove in the cutting edge, due to a defect in the enamel; this groove is deepest in the middle of the crown. The upper canines may also be peg-shaped and short, but are not characteristic.

The child soon looks old and stunted; the liver and spleen are found to be enlarged, but the lymphatic glands are atrophied or suffer slightly. There may be so-called syphilitic pneumonia of the lung, but it is extremely rare. Bones, cartilages, and even joints may show definite changes. There are often enlargements at the junction of epiphyses with the shaft of the long bones, ribs, and clavicles. There may also be gummatous swellings, but these develop at a later period: they are often symmetrical thickenings, which are often symmetrical, of the arms and legs. The joints more rarely undergo symmetrical swellings, which may subside or give rise to deformity. The knees are said to suffer most frequently.

Keratitis is frequently seen, but it often develops

Both eyes are affected at once; the cornea has a

ground-glass-like appearance, and there is marked ciliary congestion. Photophobia is present and sight is greatly diminished. This may last for months, and rarely clears up entirely, generally leaving definite opacities behind. Iritis is sometimes present.

There is much difference of opinion as to whether these hereditary syphilitics should marry. Apparently they may, and do, have perfectly healthy children.

Clinical Features of Acquired Syphilis.—The stage of incubation varies probably from 2 to 7 weeks, 5 being the average. Then the *primary* chancre or sore appears, beginning at the site of inoculation, and forming first a small papule, later a more marked swelling with flattened apex and indurated, almost cartilaginous-like base. The degree of breaking down depends on secondary causes, such as local irritation, and, in fact, the primary chancre will not develop to the same marked extent in every case, cleanliness and freedom from irritation greatly limiting its size. In most cases the primary sore remains for 2 or 3 months, and then heals, leaving a scar.

The lymphatic glands in the groin swell and become hard, but they are painless, and do not suppurate unless irritated. Sometimes the line of infection may be traced by the palpable infiltrated lymphatics.

Secondary Stage.—(1) The rashes vary in extent and degree. There is usually a roseolar rash, which comes out coincidentally with a degree of pyrexia, varying in different patients, and lasts for 2 or 3 weeks. In severe cases pains in head, back, and legs, general malaise and discomfort are present; in others these phenomena are slight or absent. (2) At the same time the throat is sore, superficial ulcers and mucous plaques appearing on the tonsils, tongue, and lips. These mucous patches are raised, moist, and whitish in colour, with sharply defined edges. (3) Condylomata or warts form most frequently about the anus or vulva, but may also be seen on the lips. (4) The skin eruptions, other than the typical early roseolar rash, have certain well-marked and classic features. They are polymorphous (by which is meant that papules, vesicles, pustules, etc., may all occur at one and the same time), coppery coloured, symmetrical in position, have a distinct tendency to form ring-like, crescentic, or serpiginous

s, and imitate many other skin affections, while they have a special predilection for the inner surface of the thighs, the perianal region of the anus, the flexor aspects of the limbs, the face, and the forehead. Occasionally pigmented areas mark the sites of old eruptions, but this is not constant. A form of alopecia is not uncommon, the hair often thinning, although it falls out in patches, while syphilitic onychia, or inflammation of the nails of the hands and feet, may occur.

Laryngeal catarrh may cause pain on swallowing and dysphagia.

a. (5) Anæmia is almost always present: the patient suffers from the toxin, but the degree varies with each case. Debility and loss of flesh accompany the anæmia. Certain writers lay much stress on the frequency of headache and sometimes trigeminal neuralgia in syphilis. These are probably connected with the anæmia and debility, and are commoner in the later stages.

b. Eye affections are very typical, and especially an interstitial keratitis which develops some weeks, at least, after the secondary stage has commenced. It is symmetrical, but one eye suffers first, and there is ciliary congestion and generally photophobia and pain. There may be retinitis or choroiditis, but these are rare. Deafness may be due to the spread of infection from the throat up the Eustachian tube.

Although a number of signs and symptoms have been enumerated, it must not be supposed that all even of those enumerated are necessarily present: sore throat and possibly a roseolar rash, with some falling out of the hair, may indicate the existence of the second stage of syphilis.

c. *Tertiary Stage* in a treated case should never appear but 6 or 8 months, and even a number of years after the primary has been acquired, tertiary symptoms may develop demanding careful attention. The third or fourth years are the most frequent periods for tertiary phenomena. In this stage, as indicated under pathological anatomy, the (1) skin lesions are deeper seated, (2) gummata develop, and in time the arterial changes due to the disease manifest themselves clinically.

There is not the same symmetry about tertiary manifestations of syphilis, and the lesions are deeper seated, and therefore serious in their effect, but they are less infective to other persons.

(1) The skin eruptions include rupia, in which limpet-like crusts cover a deep-seated ulcer, and serpiginous syphilides which resemble lupus in their appearance, but have a more crescentic margin. The polymorphous character of syphilitic eruptions may be demonstrated by other types of eruption being present as well. Sometimes a psoriasis-like syphilide develops, especially on the legs and arms, but, unlike true psoriasis, the flexor aspects do not escape. Bullæ also appear (called syphilitic pemphigus) on the arms and legs, but they become pustular, and unless carefully treated are indolent in healing.

(2) Gummata develop in many internal organs, although they may appear also in subcutaneous tissues, as, for example, under the skin of the scalp, buttocks, breasts, and many other regions, where they form painless tumours, while sometimes they develop in bursæ. They also grow in muscles, forming slightly painful, slowly enlarging tumours, which specially involve the tongue, sterno-mastoid, masseter, supra- and infrapinatus, gastrocnemius, and the rectus femoris muscles. Sometimes gummata develop in connection with the joints, and the joints may swell up, as they occasionally do in the secondary stage. The periosteum is very frequently involved by gummata, and the tibia, ulna, clavicle, and sternum are specially apt to suffer, but any bone may be affected, depending much on a preceding injury.

(3) The syphilitic endarteritis obliterans, and the less common syphilitic periarteritis, which is a development of gummata in the tunica adventitia, yield most definite symptoms in connection with the nervous system, although the coronary arteries are sometimes also involved, causing serious degenerative changes in the myocardium. It should be mentioned that syphilis may predispose to aneurism by weakening the arterial walls.

Choroiditis is a common tertiary manifestation in syphilis; the syphilitic affections of the ocular nerves will be mentioned under the diseases of the nervous system.

Tertiary Syphilitic Manifestations in the different Systems (which have not as yet been mentioned).—1. *Alimentary System*.—Gummata of the *tongue* have already been described; they may lead to ulceration. Gummata may develop in the *pharynx*, rarely in the *oesophagus* and *stomach*,

more frequently in the *small intestine, cecum, and rectum*. There is always a greater or less tendency to stenosis as the result of the cicatrization.

In the *liver* there are (1) perihepatitis, which is sometimes of syphilitic origin, and (2) gummatous hepatitis, in which nodules of varying size develop in the interstitial tissue. These nodules caseate in the centre, and tend to produce marked rigidity or cicatrization, which may be palpable, and may interfere with portal vessels or bile-ducts, causing ascites or jaundice. They are often associated with waxy degeneration. This form of hepatitis is not uncommon in acquired, but may also occur in congenital syphilis. (3) Diffuse hepatitis, which is congenital in origin, consists of an extensive infiltration of small cells really gummatous; the condition is very chronic, and the liver cells are much compressed. The organ is enlarged, and jaundice is a common result.

Hæmopoietic System.—The *spleen* is often waxy and may contain gummata. In such cases it is generally enlarged. *Lymphatic glands* are affected in secondary syphilis, as already mentioned, but even in tertiary syphilis there may be enlargement secondary to gummata in the line of the lymphatic vessels connected with the glands in question.

Circulatory System.—*Cardiac* gummata are not common, and are usually found in the wall of the left ventricle, and the heart wall may be degenerated if the coronary arteries are affected as already mentioned, and the form of degeneration is generally fibroid. Syphilitic *arteritis* has already been described under pathological anatomy.

Respiratory System.—The *nasal* syphilis so common in tertiary cases has been described. The *larynx* is frequently affected, gummata may form on the epiglottis, and there may be chondritis of the arytenoids or the cartilages of Wrisberg. Extensive ulceration may develop, and the true cords, epiglottic folds, and almost the whole larynx may suffer, the epiglottis being often affected at its free edge and on its surface. Stenosis of the glottis is a common result, often accompanied with the formation of subglottic membranes. The differential diagnosis between syphilitic and tubercular laryngitis will be described under the latter disease (p. 402), but it should be stated here that syphilitic ulceration is more painless, and the ulcerative process is very rapid. The pain of carcinoma

greatly exceeds that felt in syphilis. With prompt treatment syphilitic disease of the larynx readily yields, although for the resultant stenosis it may be necessary to dilate with bougies.

The *trachea* and *bronchi* may be the site of gummata, and perichondritis, ulceration, and eventually stenosis may be caused. This leads to marked inspiratory stridor, and, in the case of the trachea, great dyspnœa.

The *lungs* may show two types of syphilitic affection. (1) White hepatisation or pneumonia, met with in the foetus, in which the lung is in whole or part consolidated, due to a syphilitic fibrosis of the air-vesicle walls, while the arteries are markedly thickened, the lung being almost bloodless. If extensive, it is not compatible with life. (2) Gummata may form more often near the root of the lung, and associated with them is a syphilitic thickening of interstitial septa extending into the lung from the root, in which the gummata are to be found. The arteries often show syphilitic endarteritis, the neighbouring pleura may be thickened, and there is generally peribronchial thickening as well. The clinical features, such as cough and purulent expectoration, closely simulate phthisis, excepting that the lesion is chiefly confined to the root of the lung, and there are no tubercle bacilli. Syphilitic disease of the lung is rarely diagnosed in the adult, partly because it only occurs in a very small percentage of cases of tertiary syphilis, and partly on account of the absence of really typical symptoms or signs.

5. *The Genito-urinary System.*—Gummata may occur in the *kidney*, but are rare. Waxy degeneration is not uncommon, and may be associated with a form of nephritis which is possibly but not certainly of syphilitic origin.

The *testicle* is frequently involved, generally by gummatous masses, which invade the body of the organ, and not the epididymis, as in tubercle. It is painless and does not suppurate. Both testicles may suffer.

6. *The Nervous System.*—Gummata may occur, varying in size from a pea to a small tangerine orange. They are almost invariably found growing from the *meninges*, although they may invade the *brain* tissue and usually involve the cerebrum, while oculo-motor and other cranial *nerves* are often affected. Gummata in connection with the *cord* are much more rare. There may also be a gummatous development in the adventitia

the cerebral arteries called *periarteritis nodosa*. End-arteritis obliterans is a common tertiary lesion, and specially the arteries whose branches form the circle of Willis. As the result of gummata we may note the clinical features of intracranial tumour, with, in most cases, according to the author's experience, optic neuritis. Secondary inflammation is common,—encephalitis if in the brain, myelitis if in the spinal cord, while gummata, involving cranial nerves, may cause paralysis more or less complete. It is probable that patches of sclerosis develop as the direct result of the toxin or toxins. Cerebral syphilis may thus directly or indirectly give rise to intracranial tumour, a hemi- or mono-plegia, hemianopsia if the occipital lobe is involved, and paralysis of different cranial nerves, but especially the ocular ones. It may also cause a great headache and giddiness from interference with the blood-supply of the brain, and convulsive seizures of the epileptiform type are not uncommon. There is little doubt that general paralysis of the insane is often to some extent of syphilitic origin.

Cerebral syphilis is similar in that gummata and endarteritis are present, although less frequently than in the brain. Usually spinal meningitis is not uncommonly of syphilitic origin, and disseminated patches of sclerosis in the cord may be due to the syphilitic toxin. It should also be repeated that locomotor ataxia, spastic paralysis, and other cord lesions, in some cases be due to syphilis. It is most important for the clinician to remember the possibility of syphilis, to try to obtain definite history from the patient, and in every doubtful case to try antisyphilitic treatment. Sometimes other evidences of syphilis may be noted, which are either typical in themselves or support a syphilitic hypothesis.

Diagnosis of Syphilis.—Many patients refuse to admit to a syphilitic history, and we may require to look for evidence of a primary or of secondary or tertiary lesions. A history of the hair falling out, with sore throat and skin rashes, suggests secondary syphilis, and the deeper-seated tertiary lesions leave scars. When repeated miscarriages are very common, due to placental degeneration. Examine the tibiae for nodes, the eyes for iritis or choroiditis, and note if the testicles have been enlarged.

Antisyphilitic treatment is so successful in specific cases

that a diagnosis may only be definitely arrived at after a prolonged course has been carried out.

Prognosis.—In most cases a favourable result is assured if treatment has been carefully, thoroughly, and conscientiously attended to. In untreated patients tertiary manifestations are very apt to cause permanent damage, and especially stenosis of some important structure, or where in the brain gummata or advanced syphilitic arteritis are present.

Treatment.—It is unnecessary to enter into a lengthy discussion of the prophylaxis of syphilis. Continence is possible, and, as Professor Osler along with many others has wisely remarked, hard work, physical and mental, does much to keep the lower side of human nature in check.

The question of State regulation of vice has caused many stormy scenes. Unfortunately the transgressor of the moral laws in question is not the only sufferer, for he may hand down a heavy load of misery to his children. Syphilis cannot be considered cured short of a period of 2 years from the last appearance of the rash or other manifestations, and therefore marriage, before that period has elapsed, is an inexcusable offence; but with all the care possible a recrudescence of syphilis may appear even after that period has passed. It is, however, a certain satisfaction to know that tertiary lesions are not nearly so infective.

For the primary chancre, cleanliness is the only treatment. The knife is rarely resorted to for the purpose of excision, because the futility of the operation is evident, the virus passing through the body at a rapid rate and being in no sense limited to the primary site of inoculation.

For the secondary manifestation, mercury is the great remedy, and it may be used in several different ways, but it is wise to get the teeth overhauled beforehand by the dentist, and everything done to keep up the general health.

(1) *Internal Administration.*—Hydrargyrum cum creta may be ordered in small doses (gr. 1-2), with a similar quantity of Dover's powder, 4 times a day. The liquor hydrargyri perchloridi may be taken internally in drachm doses (gr. $\frac{1}{8}$) thrice daily, or the biniodide of mercury in the same dose may be taken in pill form. Such treatment, begun early and steadily persevered with for months, is most efficacious in aborting the secondary stage. Some authorities give mercury for 2 months,

1 month without, then 2 months with and 3 months without, so that in 2 years the patient has 10 months' treatment and 14 months without (Fournier). In the later years 4 courses of mercury are given—4 courses in the 3rd year, and 3 courses in the 5th year. Other authorities, as Hutchinson, give long continuous treatment. Guard against salivation by careful attention to the teeth and by using astringent mouth-wash, and check diarrhœa by careful diet and opium.

Inunction is another favourite method, the ointment being the unguentum hydrargyri—being rubbed into the skin eight or ten times a day, morning and evening, and about 40-50 grains of the ointment at a time. Rub for 30 minutes, and, if the rubbing is performed by the patient himself, the hands should be washed. The sides of chest and abdomen and the inner thighs are often selected, and the area of application should be changed each time. Mercurial soaps are often used, the lather being well rubbed in and left to dry. The treatment may be dropped one day out of seven.

Fumigation.—The patient sits on a chair covered with a cloth so as to keep in the fumes. About 20-40 grains of mercuric iodide are volatilised in a small capsule over a flame, and steam of water is also used, so that the mercury is absorbed in a mild steam bath. About 20 to 30 minutes should be allowed for the application.

Hypodermic injections may be given deeply into the skin, either the perchloride or biniodide being preferred (the former is preferred); intravenous injections have been tried, but are not recommended. Care should be taken with intramuscular injections to avoid the risk of local necrosis, due generally to the injection being too superficial. The injections are only given once every day, or every two or three days, depending on the dose administered.

In infants and children, the subjects of hereditary syphilis, inunction is one of the best methods of administering mercury, and the ointment may be applied on the child's back. Hydrargyrum cum creta is another favourite in doses of 5 grains given internally thrice daily, depending on the age of the patient.

For the treatment of tertiary manifestations of all kinds potassium iodide is administered, beginning with 10 grains, thrice daily,

and increasing until twice or thrice that dose is being taken. Attention to the bowels and general health is usually sufficient to prevent iodism with its resultant sore throat and severe coryza, but there are patients who cannot take it. Sometimes doubling the dose lessens iodism; still in certain cases it is wise to stop treatment from time to time.

For syphilis involving the internal organs, besides the specific treatment just mentioned, symptoms such as dropsy, headache, etc., may call for our help.

Carefully regulated diet and a minimum of exercise are advisable, and the best holiday for a syphilitic patient is a sea voyage. Marriage, as already stated, should be discouraged for long after the period of infection, and should be absolutely forbidden until 2 years after the last obvious symptom has disappeared.

It is doubtful to what extent syphilis, properly treated, shortens life, although neglected syphilis certainly has every chance of so doing. Many insurance offices add a few years to the age of the aspirant for insurance, others defer the petition until all trace of the disease has disappeared.

XXXVII. GONORRHOEAL INFECTION

WHILE gonorrhoeal rheumatism is most important because of its more frequent occurrence, it must be remembered that the gonococcus may produce a general gonorrhoeal infection as well as a localised involvement of joints.

Etiology.—The gonococcus infects mucous membranes, and is usually acquired by sexual intercourse, although the conjunctivæ, as in new-born children, may be the primary site of infection. The organism has been found in the blood.

Pathological Anatomy.—The gonococcus sometimes finds its way up the urinary tract, or by way of the Fallopian tubes it may infect the general peritoneal cavity. In the joints infected by the gonococcus there is an effusion, rarely purulent, but containing the organism, and there is often inflammation in the sheaths and tendons, and in connection with bursæ, while the conjunctivæ may also be involved.

1. *A General Gonorrhoeal Infection.*—In this condition the patient usually dies of acute gonorrhoeal septicæmia, and often

apidly. Occasionally there are numerous pyæmic foci and throughout the body.

Gonorrhœal rheumatism is more common in men than in women, and generally occurs before middle life. There are many types of the affection which vary considerably. Many large joints may be involved, and often the smaller joints. In other cases only a few joints alone suffer, while sometimes a single joint is affected. One peculiarity of gonorrhœal rheumatism is that certain joints suffer in gonorrhœal arthritis which, as a rule, do not escape in other forms of rheumatism. These are the hip-joint, the maxillary, the sacro-iliac, the sterno-clavicular, and the cervical. The effusion does not, as already stated, usually become purulent, but the affection is long standing, and does not yield satisfactorily to treatment.

Gonorrhœal bursitis and gonorrhœal involvement of the ligaments and fibrous tissues are not uncommon, either associated with arthritis or distinct from it.

Gonorrhœal conjunctivitis is generally the result of direct infection, and may cause a severe iritis, possibly with ultimate loss of the eyeball.

Prognosis is favourable as regards life, in most cases, even in general gonorrhœal infection; but gonorrhœal infection is always tedious and may cause great and permanent deformity.

Treatment.—Potassium iodide is certainly a remedy which should be tried, but salicylates are useless. Tonics such as iron, arsenic, and cod-liver oil are invaluable. Locally, treatment may be done by hydropathy, and in some successful cases the affected joints have been incised and washed out with antiseptic lotions.

XXXVIII. TUBERCULOSIS

There is no disease so wide-spread or so fatal to the human race as tuberculosis. It is specially common in the large cities, but is much less frequent in country districts. Fully 11 per cent. of the total mortality is due to one or other of the forms of tubercular disease.

Tuberculosis is to be found in all countries, but is certainly

less prevalent in the tropics than in more temperate regions. At high altitudes where, owing to the excessive purity of the atmosphere, there is less risk of propagation of the organism, tuberculosis is rare, and were it not for the number of phthisical patients who frequent the Alpine health resorts, the local death-rate from the disease would be very low. Little can be said with regard to racial tendency to the disease, because it depends more on the sanitary conditions of home-life than on any difference as regards nationality. Tuberculosis has greatly diminished during the past decades, and there is no disease with regard to which such satisfactory progress has been made, due more to sanitary reform than to any new medicinal treatment,

Tubercle bacilli, discovered by Koch in 1882, are short rods $1.5\ \mu$ to $3.5\ \mu$ in length (a red blood corpuscle is about $8\ \mu$) and $.3\text{--}.5\ \mu$ in breadth. They are slightly curved, and vary in length according to the age of the culture. Some show clear, refractile spaces which do not stain. These are not spores, and by many authorities are thought to be signs of decay. Sometimes the bacilli occur in threads, and occasionally they have delicate, branching processes, which are cultural peculiarities.

The **staining** method usually employed is the Ziehl-Neelson method—fuchsin grm. 1, phenol 5 per cent solution in distilled water grm. 100, absolute alcohol grm. 10. This should be raised to the point of steaming, with the cover-glass, film-side downwards, floating in it, and the cover-glass must be left in the stain for 2 minutes. Then, after washing with water, decolorise in a 20 per cent solution of sulphuric acid and water; then wash in weak alcohol, and counterstain in a weak aqueous solution of methylene blue. The bacilli are acid-fast, which means that they retain the fuchsin in spite of the action of the sulphuric acid—a peculiarity which they possess along with a very few other organisms, such as the leprosy bacillus, timothy grass bacillus, smegma bacillus, etc.

Cultures may be made on blood agar, glycerin agar, or potato, and are kept at blood-heat. They grow slowly, and the colonies are greyish-white in colour.

There is no question of the existence of differences both in form and also in virulence of the tubercle bacillus.

The Virulence of the Tubercle Bacillus.—Many patients

ng from acute pulmonary tuberculosis spit millions of le bacilli in the daily expectorations. Dried sputum is virulent for months, although after a time it gradually ts infective power. The chief danger is unquestionably d sputum dust getting into the air, and dust which may i the tubercle bacillus. Bacilli retain their virulence veral months in water, and freezing has no effect in ing the organisms. Moist heat is more destructive ry heat, and dried bacilli survive in a considerably temperature. Most authorities agree that at 70° ade the tubercle bacillus can be killed in 5 to 10 s. Sunlight is one of the best natural sterilising agents bacilli. It destroys them in a period varying from s to hours, depending on the strength of the solar rays e thickness of the infective material to be acted on. re to the atmosphere apart from the sun kills the vithin a week.

bolic acid solution of 1 in 20 and corrosive sublimate 1000 may be safely depended on for the purpose of ng tubercle bacilli.

Toxic Products of the Tubercle Bacillus.—It is possible to discuss here the various toxic agents which en separated from the tubercle bacillus by different

The dead bacilli have been found capable of stimu- localised inflammation round about tubercular foci, and tly in this way that Koch's tuberculin acts.

Distribution of the Bacilli.—The bacilli are found r or less numbers in all tubercular lesions, although which are markedly caseous the number present may small. There is a tendency for the local spread of li chiefly by the lymphatics, although if the organisms e blood-stream, they may be diffused widely through- body. Pathologists are agreed that tubercle bacilli ain for many years quiescent in an old tubercular d that, given the opportunity, they may then multiply, se foci may become once again active centres for sion of the disease. The urine frequently contains cases where the kidneys are involved, but milk and ve not been proved to be infective unless there is a r lesion of the nipple or breast.

Histology of the Tubercle Nodule.—There is

nothing really distinctive in the tubercle nodule apart from the presence of the specific organisms.

According to Baumgarten the *tubercle bacilli*, having reached the focus where a tubercle nodule is about to develop, proliferate, and this is followed by a multiplication of connective-tissue cells, endothelial cells of blood-vessels, and possibly other cellular elements, all of which may form the typical *epithelioid cells*.

Numerous *lymphocytes* now appear, not merely in the nodule, but also round its periphery, and polymorphonuclear cells accumulate also in the neighbourhood of the nodule.

There is invariably a *reticular matrix* or network, which acts as a supporting structure, and which probably develops from connective tissue.

Lastly, in many tubercle nodules *giant cells* may be present. These are generally formed from fusion of several epithelioid cells together, and nuclei may be seen round about the periphery of the cell. Giant cells are more frequently present in older nodules, and in them, and also in the epithelioid cells, tubercle bacilli are often found.

A tubercle nodule tends to caseate because it is extravascular, and the process is really a coagulation necrosis. Very often in place of, or secondary to, caseation, fibrous tissue replaces the degenerating nodule, or calcareous deposition occurs in the caseous material.

Not infrequently tubercle nodules run together to form larger tubercular masses, and this process may be well studied in tuberculosis of the lung. Many secondary changes may be induced in connection with tubercular nodules, such as a localised catarrhal pneumonia round about the tubercular foci in the lung, and often suppuration.

Modes of Infection.—1. *Intra-uterine Infection.*—So far as we are aware, (a) spermatozoa do not carry infection, and very rarely at any rate is (b) the ovum attacked by the tubercle bacillus before it reaches the uterus: (c) placental blood may certainly transmit the infection to the fetus. Probably there are relatively few cases of intra-uterine tuberculosis, hereditary predisposition being rather evidenced in later life by the phthisical type of chest and a lessened power of resistance to the tubercle bacillus.

2. *Inoculation.*—The fingers may be inoculated from in-

the meat or pus, and the verruca necrogenica of pathologists is an example. Butchers and others exposed to tubercle in dealing with tuberculous carcasses are also liable to infection, and washerwomen may suffer from washing infected linen. Opponents of vaccination cite a few cases where tubercle has been inoculated with the lymph.

Inhalation.—Dried sputum containing tubercle bacilli, either on floor, handkerchiefs, clothes, or beard and moustache, readily set free in the air numerous organisms, and dust of the floor in a sick-room inhabited by a consumptive patient often contains many bacilli. Inhalation is usually the chief mode of infection, and persons in attendance upon consumptives who are careless and dirty in their habits are often themselves infected.

Infection by Meat and Milk.—Meat is not generally undercooked in this country, and, besides, tuberculous meat is condemned by the authorities and destroyed, but cows suffering from tubercular disease of the udders give infective milk, and the frequency of involvement of the tonsils, cervical and mesenteric glands in children, is most easily explained by the hypothesis of an infected milk-supply.

It is impossible to discuss Koch's theory that bovine and human tuberculosis are distinct as regards the organisms responsible, but until his statements are placed beyond possible doubt it is desirable to err on the safe side, and to consider both meat and milk alike capable of infecting the human subject.

Conditions which predispose.—Those who live in stuffy, ill-ventilated dwellings and work amongst insanitary surroundings are much more liable to be attacked, and unquestionably soldiers, sailors, and prisoners who are kept in close confinement are all victims to the bacillus in large numbers, the proportion of deaths among such persons due to tuberculosis is proportionally very high. In the past decade the fall in the death-rate from tubercular disease has improved 50 per cent owing to sanitary and hygienic reforms.

There is a "tubercular" constitution, or, more correctly, a hereditary predisposition. These persons have typically ill-developed chests, alar, flat, or long-shaped, and often a combination of all together, and they have a lowered vitality which renders them less resistant to the attack of the bacillus. Two types of face are common in such persons. The one type are

small, delicate-featured, with fine skin, short upper lip, long eyelashes, and they are usually of short stature. The other type have irregular features, with coarse skin, thick lips, and are clumsy and frequently tall in stature. These two types are very often seen among those who fall victims to tuberculosis.

Age.—While tubercular disease is met with at all ages, pulmonary tuberculosis is most frequent between 20 and 40, and in young children the alimentary system is more apt to be the site of involvement than it is in the adult.

Occupations.—Many trades which are responsible for dust diseases predispose, and persons exposed to sudden changes from great heat to cold, or whose occupation necessitates getting wet, are specially susceptible.

Disease.—Many lung diseases predispose, but especially pleurisy, often in itself tubercular. In diabetes mellitus death is frequently due to a secondary pulmonary tuberculosis, and in any heart affection, such as pulmonary stenosis, in which the lung suffers from a deficient supply of blood, there is a tendency to tubercular infection. All debilitating diseases may be said to predispose. Excessive lactation certainly is injurious, and in tuberculous women pregnancy, while there may be a temporary arrest of the disease, is generally followed after parturition by a rapid advance.

Treatment. — All the important tubercular affections referable to the different systems are described under the diseases of the systems in question, and in connection with most of these the diagnosis, prognosis, and treatment are discussed. It only remains, therefore, to refer to the tuberculin treatment of tuberculosis and the more recent modification of that treatment, so far as it is possible to speak with certainty. The old tuberculin originally devised by Koch had been largely given up, but either the original preparation or modifications of it have received more attention recently.

Professor Wright of Netley recommends the use of Koch's tuberculin T. R. in order that the patient's leucocytes may acquire a greater capability for the destruction of tubercle bacilli by phagocytic action. The procedure is as follows :—

The opsonic index or phagocytic index of one's own leucocytes for dead tubercle bacilli must first be ascertained. Serum-free corpuscles are obtained by washing the blood in

mixture of 1 per cent citrate of soda in normal saline solution, and later in normal saline solution alone. Then mix suspension of dead tubercle bacilli with the blood corpuscles of the serum of the individual who is to be examined. Incubate the mixture at a temperature of 37° Centigrade for 24 hours. Prepare films, and stain by the Ziehl-Neelsen method, counting the number of tubercle bacilli in each field. As a general rule about 5 bacilli will be found per each leucocyte, and this may be taken to be a normal opsonic index, or equivalent to 1.

In the case of the tubercular patient his serum is used instead of that of the normal subject, because it is upon the serum that the test depends, and where the patient is suffering from chronic tuberculosis there is a marked fall below normal opsonic index, often reaching .5, .6, or even lower.

It has been found that the administration of tuberculin often raises his opsonic index greatly above normal, and that this is associated with a distinct local improvement in the tubercular condition.

In acute cases, however, the opsonic index is almost always much above normal, and therefore there is little use of tuberculin injections. Care must be taken, as recently pointed out, to test the opsonic index of patients, either while resting, or always after a definite amount of exercise, there is a rise in the index associated with exercise.

The administration of fresh doses of tuberculin should be timed with the rise in the opsonic index of the patient, usually described as the positive phase, and the rise or fall phase follows a fall or negative phase, the latter resulting from the injection of tuberculin. Inoculating during the negative phase, or too soon for the case in the positive phase, might still further lower the opsonic index. It is therefore treating a case, to begin with small doses of tuberculin, and to exceed $\frac{1}{500}$ milligramme.

XXXIX. MALARIAL FEVERS

of fevers, which may be benign or malignant in which the parasite occurs in the red blood corpuscles, producing anæmia and changes in the spleen, liver, and

other organs which are mainly the result of the deposition of blood pigment.

Malarial diseases depend for their existence on a particular kind or kinds of mosquitoes, and consequently warm climates, associated with marshy ground in which mosquitoes can breed, are essential factors. The mystery surrounding malarial fevers has been elucidated by the invaluable work of Laveran in 1880, and later by the investigations of Ross in 1898, and others, with regard to the *rôle* which the mosquito plays in connection with the disease. Different types of malaria are met with in different countries, varying considerably in their severity.

Etiology.—As already stated, marshy ground tends to increase the mosquitoes which act as the intermediate host for the parasites. Unquestionably the natives of a country suffer less markedly than those who are only temporary residents, and persons before middle life are apparently specially liable to attack.

BENIGN MALARIA USUALLY INTERMITTENT IN TYPE

The Parasites.—There are two forms of parasites which may occur in this fever: the one parasite is the tertian, the second is the quartan. These terms mean that the organism sporulates every 48 hours in the tertian form, whereas in the quartan sporulation occurs every 72 hours. There may be two groups of tertian parasites, so that the patient may have sporulation occurring every day; and similarly there may be three groups of quartan parasites, so that it is possible to have a daily attack of fever where the quartan parasite is present. This is called *quotidian ague*. The possession of several groups of organism implies that the individual has been several times infected by mosquitoes, constituting double tertian, and double or triple quartan, as the case may be. It by no means always happens that two or more series of parasites sporulate at an interval of a day, because two groups may sporulate on the same day, and then a day may pass without any attack of fever.

The Tertian Parasite.—These are small, colourless, somewhat ring-like bodies, with a distinct amoeboid-like movement, and generally there is at one part of the ring a small quantity of pigment. These colourless bodies increase in size, and the

blood corpuscles, in which they are, become swollen and the hæmoglobin of the corpuscle being utilised by the parasite for the production of the pigment granules. The granules of pigment show rapid movement, and they eventually occupy a central position. Towards the end of the 48 hours the pale-looking parasite becomes divided into 12 to 24 segments, and when this segmentation has occurred the parasite is termed sporulation. These segments or spores are free in the blood-stream, whenever the red blood corpuscle forming the host breaks up, and at the same time the pigment granules escape also. The spores are now free to infect a fresh series of red blood corpuscles and so perpetuate the succession of attacks of the fever. This sporulation or segmentation occurs almost exactly at the end of 48 hours, and co-existent with the sporulation is the attack of fever.

The Quartan Parasite.—In this case the original ring-parasite shows at first little difference from the tertian form, only the amœboid movements of the parasite are absent, the pigment granules are coarser and darker in colour, and the segmentation gives rise to the development of 6 to 12 segments or segments in place of 12 to 24. The sporulating and quartan parasites are the asexual organisms, and after a period of 7 or 8 days sexually mature organisms develop, which are the micro- and macro-gametocytes described in the further stages under the extra-corporeal cycle in the malarial protozoa.

ANOTHER TYPE OF MALARIA, SOMETIMES CALLED ÆSTIVO- TROPICAL FEVER, WHICH IS GENERALLY REMITTENT IN TYPE

This group includes the comparatively mild types of æstivo-tropical malaria met with in Italy near the Maremma, and also includes very malignant forms of malaria, such as those which have proved so fatal on the West Coast of Africa and elsewhere. In the milder cases the presence of the parasite is definitely made out, and the patient suffers rather from the attacks, with headache, and enlargement of the spleen. In the course of time considerable anæmia develops: but such attacks of fever may undermine the patient's health just as much as the more obvious benign or intermittent varieties.

already mentioned. Other cases belong to the pernicious or malignant forms of malaria, but the whole group is described as malignant, and fully deserves that title.

The Malignant or so-called *Æstivo-Autumnal* Parasite.—Different descriptions have been given of the organisms concerned, although they may in reality be one organism whose virulence is modified by certain conditions. They are, (1) the pigmented quotidian, (2) the unpigmented quotidian, (3) the malignant tertian: in all these organisms there is a much more indefinite period at which sporulation occurs, and in the peripheral blood there may be little evidence of the parasite at all. The parasites are chiefly confined to the internal organs, especially the spleen and the marrow of the bones, in which, however, they are invariably found. There is nearly always the typical ring-like parasite with little pigment, and generally marked changes in the red blood corpuscles which form their hosts, in the direction of wrinkling of the corpuscles and rapid disappearance of hæmoglobin. In their life-history, however, the intra-corporeal parasites, although they differ in certain details from those belonging to the intermittent group, still show a family resemblance to them, and outside the body the mosquito plays the same part as with the intermittent organism.

Crescent-shaped bodies are distinctive of the malignant or *æstivo-autumnal* parasite. It is crescent-shaped, and the pigment may be either diffused throughout the parasite, or may appear as a central accumulation. It is the sexually mature form of the parasite referred to later.

Staining Methods.—A blood film, prepared in the usual way, should be taken 8 to 12 hours before sporulation is due. Fix the film with ether and absolute alcohol in equal parts, or with absolute alcohol alone, then stain with Romanowski's or Leishman's modification of Romanowski's stain. The parasite is coloured blue, and should be examined with an oil-immersion lens, by preference $\frac{1}{2}$ th or $\frac{1}{4}$ th inch objective.

EXTRA-CORPOREAL PHASE OF THE MALARIAL PARASITE, OR THE CYCLE OF DEVELOPMENT IN THE MOSQUITO

Ross, Manson, and many others discovered that it is by the mosquito, and the species called *anopheles*, that the

rial parasites are taken up from the blood of the human subject, and go through a stage of development entirely distinct from what is noted in the human subject.

The parasites of benign tertian and quartan ague, and the malarial types of parasites, occur in sexually mature forms in the human subject. These forms do not sporulate. The tertian and quartan mature, non-sporulating parasites are of two kinds—the hyaline or male organism called the micro-gametocyte, and the granular or female organism called the macro-gametocyte. In malignant types, due to the so-called malarial parasite, the crescentic bodies are the sexually mature forms.

No further change occurs in the human subject, but the process of impregnation of the female parasites by the male can be carried on a wet slide, and the further cycle of development takes place in the stomach wall of the mosquito.

The male parasite, or micro-gametocyte, develops flagella, and micro-gametes, which move vigorously, become free from the parent organism, and seek out the macro-gametocyte or female organism. One enters at a papilla which the female organism projects, and after moving vigorously inside the cell disappears from further observation. The macro-gametocyte is then fertilised, and is called a zygote; this undergoes a change in shape, one end becoming pointed like a spear, which is the zygote, now called an ookinet, to pierce the stomach wall of the mosquito. It reaches the muscular coat, and there it becomes encysted about the second or third day, and develops into an oocyst. This grows rapidly, and may attain a diameter of 80 μ . Inside the oocyst the small slender sporozoites develop in large numbers, and eventually the parent oocyst bursts; the sporozoites escape, and a certain proportion of them migrate to the veno-salivary gland, ready to infect the next mosquito which the mosquito may bite.

The crescentic bodies of the malignant types undergo a similar cycle in the mosquito.

It is easy to distinguish the anopheles mosquito from the culex because the body of the anophele is placed at right angles to the surface to which the animal is attached, whereas the body of the culex is parallel with the surface. The wings of the anopheles are spotted, and the palpi are also distinctive. The anopheles require a special kind of algae in the water in

which they breed, and consequently they are not so wide-spread as are the culex mosquitoes. In addition anopheles require not merely sheets of water, but also a steady warm temperature.

TYPE I. BENIGN AGUE USUALLY INTERMITTENT IN TYPE

Clinical Features.—There are three stages which are associated with the sporulation of a group of parasites.

1. The *Cold Stage*, during which the patient shivers, looks blue and cold, suffers from violent rigors; associated with these symptoms are headache, extreme languor, and often yawning and nausea. Notwithstanding the fact that the skin

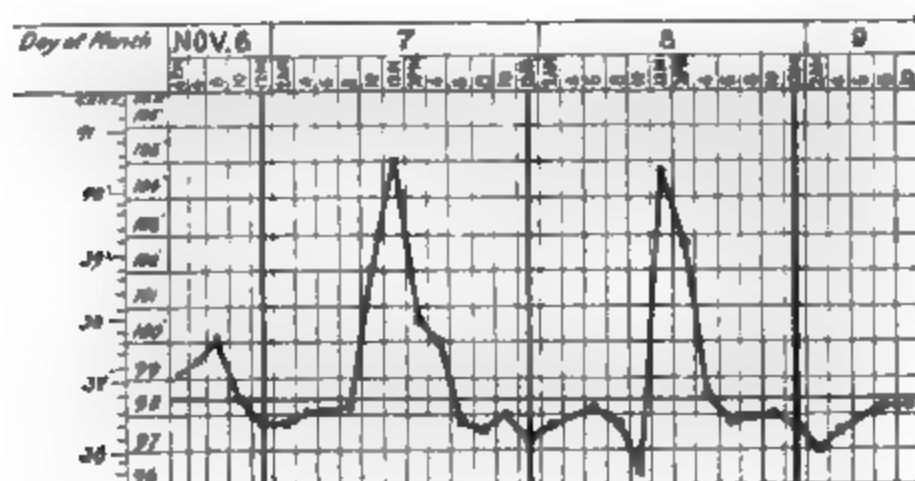


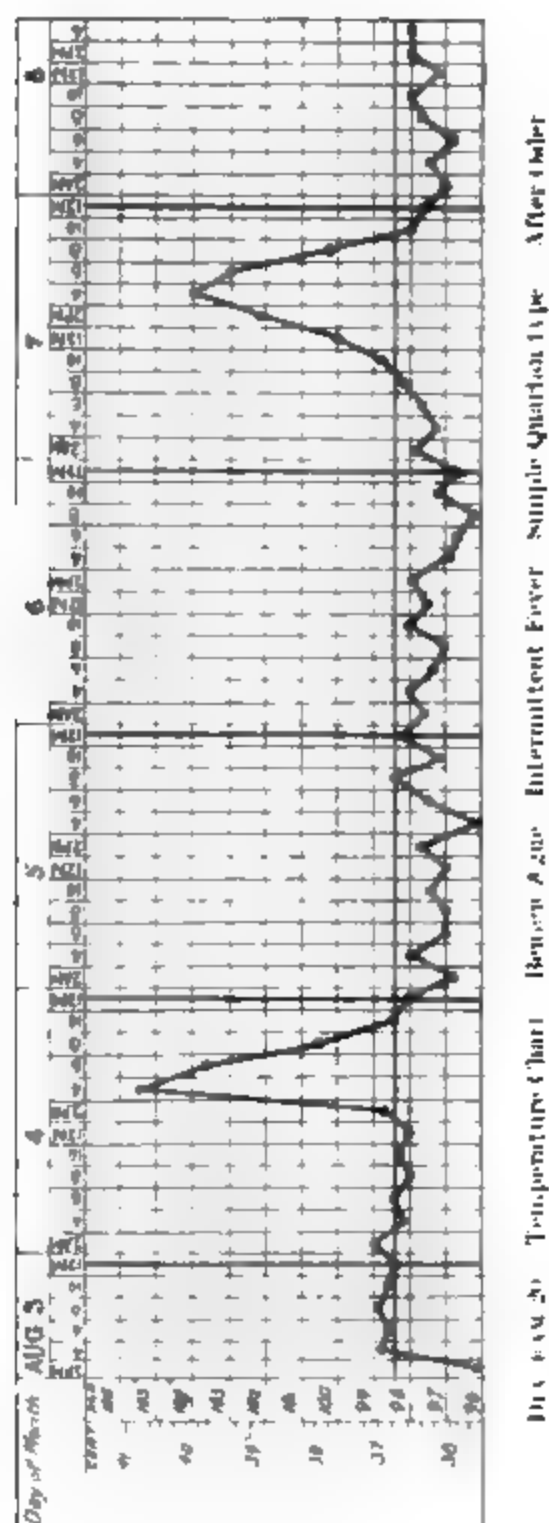
DIAGRAM 19. —Temperature Chart. Benign Ague, Intermittent Fever; Double Tertian or Quotidian. After Osler.

temperature is reduced, the temperature in the rectum and the mouth will be found to be 103°, 104°, or 105° F. This stage lasts for some minutes up to about an hour.

2. The *Hot Stage* follows the cold. The patient soon feels warm and comfortable, and throws off the blankets with which during the preceding stage he has covered himself, but in a short time he begins to burn, his head throbs, and his pulse is full and bounding; he complains of thirst and severe headache, pains in the back and legs, not infrequently an element of delirium is superadded, perhaps especially in children. This stage lasts generally for a period of 1 to 2 hours, and is followed by—

3. The *Sweating Stage*, during which the temperature falls with profuse sweating, and the discomfort rapidly subsides. The duration of this stage varies; it may last for 1 or 2 hours.

he same time, almost exactly
attack, unless there happens to



severe, and the term "bilious
uch cases.

ns of malaria are instances of
e remembered that a pernicious
ntermittent fever as well as in
n the severity of the attacks,
ineffective resistance of the

There are certain types of malaria more common in the remittent than in the intermittent forms, about which a few remarks must be made.

The Bilious Remittent Type.—In this form of malaria there

is great alimentary disturbance, with specially marked epigastric pain, enlargement of spleen and liver, and much bilious vomiting. Sometimes diarrhoea occurs, although more often constipation. It may be a stage in remittent or intermittent fever, and the alimentary phenomena tend to become less marked after a time.

The Typhoid Type.—

Some cases closely resemble typhoid fever from an early stage, and the clinical features designated "typhoid" are the result of excessive weakness, with usually a tendency to a low type of delirium or coma, a feeble pulse, a dry tongue, and frequent picking at the bedclothes—a group of symptoms often described as "typhoid" in connection with any febrile process. The typhoid type of malaria is, however, extremely dangerous; any malarial fever may pass into a condition of things which can be described

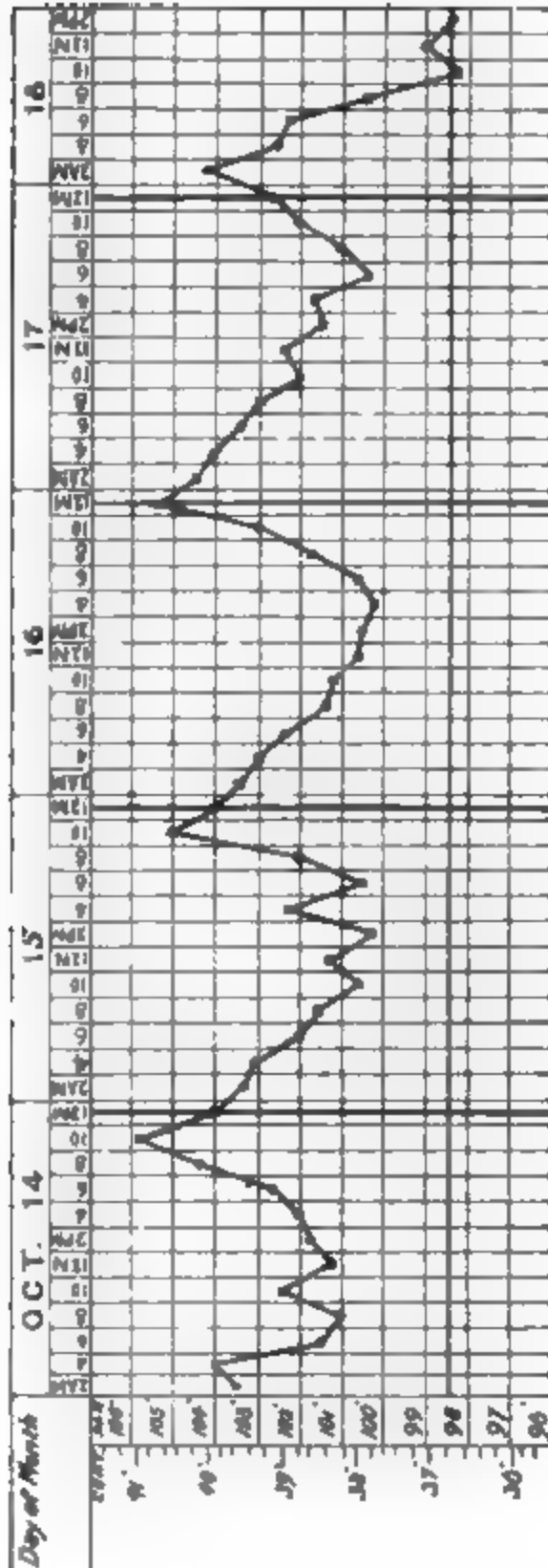


DIAGRAM 21 Temperature Chart Malignant Ague, Remittent Fever, Quotidian attack.

only as "typhoid," and bedsores, a low type of pneumonia, or sheer debility very commonly lead to a fatal termination of the case.

pernicious Malaria.—This also is more common in recurrent fevers. It is generally held to be due to the blood-vessels being plugged by thrombi consisting mostly of pigment, and in part of parasites. The results can easily be varied; there may be (1) head symptoms, sometimes with convulsive seizures, often with more acute delirium and a tendency to hyperpyrexia, while in certain cases convulsive seizures occur, and death only too frequently follows in 12 or 24 hours.

(2) In other cases it seems as if the thrombi involved the intestinal tract. Profuse diarrhoea, with intense weakness and cholera-like cramps, soon places the patient in a very bad condition. In some of these cases the diarrhoea is dysentery, others more closely resemble cholera, and in others the condition previously described by the term "typhoid" speedily develops.

A third variety of pernicious malaria is well described under the term "hæmorrhagic." In it blood may flow from any surface, and not infrequently the urine contains, not red corpuscles, but hæmoglobin. It is a paroxysmal hæmoglobinuria, and when it is associated with vomiting of large quantities of blood and with bleeding from the nose, and elsewhere, the term "blackwater fever" has sometimes been applied to it. This is the fever which we meet with the malaria of West Africa, and especially the East Coast, although it has been met with in many other countries, and it should be remembered that any fever may assume a hæmorrhagic type under conditions which are as yet inadequately explained. It has been suggested that quinine is responsible for some of these cases, but as yet there is no proof that this is correct, and though it is not, as a rule, looked upon as such an efficacious remedy in these cases, its administration should not be withheld.

Malarial Cachexia.—When an individual has experienced repeated attacks of malarial fever, he becomes cachectic. By this is meant a profound anæmia, due to the destruction of large numbers of red blood corpuscles, and, in addition, a tendency to the accumulation of pigment in different parts of the body. Certain of these cases eventually become chronic or malignant in type, but in others the patient

simply suffers from the prolonged loss of red blood corpuscles, and his symptoms include those of severe anæmia with breathlessness, œdema of the legs, and huge enlargement of the spleen, which remains permanent, and to which the term "ague-cake" has been given. Added to these phenomena are not infrequently hæmorrhages, a constant tinge of jaundice, and a tendency to scorbutus. The hæmorrhage may be retinal, or from the nose, gums, mouth, and elsewhere, and the patient's skin acquires a typical brownish or greyish colour.

A number of cases of so-called malarial cachexia have recently been found to be really due to the presence of Leishman-Donovan bodies in the splenic blood. These organisms are closely related to trypanosomes, and are believed to be the cause of kala-azar.

Not infrequently the clinical features in malaria suggest the special involvement of some particular system,—occasionally, for instance, severe sickness and vomiting are prominent, while in other cases headache and neuralgia of a very severe type constantly harass the sufferer.

It should be remembered in connection with all malarial fevers that the disease may simulate many other conditions—vomiting, dysentery, pleurisy, pneumonia, severe palpitation, syncopal attacks, apoplectiform seizures, and paroxysmal neuralgias may all develop along with, or as part of, a malarial seizure, and in the malarial subject the possibility of part, at least, of any ailment being of malarial origin should never be overlooked.

Malarial attacks may last for a long time. They may, apparently, be cured in two or three weeks, but relapse is extremely common, and it is prudent, when possible, to induce the patients to change their place of residence to a country in which malaria is unknown, because repeated attacks of malaria induce malarial cachexia.

Diagnosis.—Even in the remittent forms of malaria there is a great tendency to periodicity, and in all forms of malaria the spleen is enlarged. During an attack, or rather just before sporulation, the blood should be examined for the parasite, and the remarkably beneficial effect of quinine in preventing attacks is an important point in the diagnosis.

Prognosis.—From the previous pages it will be gathered that malaria may be mild, or may be extremely fatal, and that

remittent fever is more serious as a general rule than intermittent. Perhaps still more depends on the possibility of the individual attacked residing for the future in a non-malarial country, but there are many persons who stave off malarial seizures by carefully regulated doses of quinine. The malarial fevers acquired in West Africa are very commonly of a serious type.

Treatment.—**PROPHYLAXIS.**—Long before the relationship of the mosquito to the malaria parasite was understood, it was known that marshy ground predisposed to malaria. Certainly the draining of swamps where the anopheles can breed is of the utmost moment, and where infected mosquitoes exist, the mosquito curtain should invariably be used during the night. Much has been done by pouring kerosene oil over the surface of pools which cannot be easily drained, in order to destroy the larvæ of the mosquito. It is wise, when an individual has to live in a malarial country, not merely to attend to the mosquito net, and to free the district immediately surrounding his home from mosquitoes by the use of kerosene oil and drainage, but also to avoid exposure in the evening when mosquitoes are apt to bite. Furthermore, after exposure in a specially malarial district, he should take a dose of quinine, sometimes 5, sometimes 10 grains, the amount depending on the personal equation of the patient.

It is best to give quinine, and to give it promptly, in treating ordinary cases of malaria. Ten grains must be taken at once, and then smaller doses of 4 or 5 grains thrice daily should be kept up for several days. It is well to endeavour to abort the periodic attacks of fever, in other words, to anticipate sporulation by giving quinine; and it should be remembered that in order to have the full effect of the drug, it must be administered on an empty stomach, and preferably with a little dilute sulphuric or hydrochloric acid, so that the quinine may be dissolved and thus be given every chance of acting. Much trouble, however, is experienced by the great tendency in many malarial patients to sickness and vomiting, and in these cases quinine may be given as an enema or even hypodermically,—the latter method, however, being undesirable, because an abscess not infrequently results unless great care is taken. Hypodermically the hydrochlorate of quinine ($7\frac{1}{2}$ grains thrice


daily) is recommended by Manson. The injection should be made deeply; but as far as possible avoid the use of this method of administration altogether, because in several cases tetanus has supervened notwithstanding every possible care being taken.

Calomel and ipecacuanha are often of advantage in allaying the vomiting of malaria, while ice and effervescing mineral waters should not be forgotten. During the cold stage of an ordinary attack of intermittent fever give the patient hot drinks, apply hot bottles to his feet, and wrap him well up in blankets. During the hot stage sponge the skin and apply an ice-bag to the head, and when the sweating has commenced guard against chills, and during this stage give a dose of quinine.

It is not desirable, where there is a very high temperature, to give antipyrine, or one of that group of remedies, but rather to resort to cold affusions, the application of ice to the body, and similar methods. For hæmorrhagic cases quinine should not be neglected, but calomel has proved of great value given in full doses, and tannin and other astringents are also commended. Never forget that malaria means anæmia, and that iron and sometimes arsenic are beneficial, while careful dieting and change of air and climate will do much to restore health.

The enlargement of the spleen is extremely difficult to treat satisfactorily; where it is painful, hot applications and certainly the use of quinine internally do much to reduce the size of the organ, but where ague-cake is present, no treatment seems to be efficacious.

There are many other remedies recommended in various countries for malaria, and methylene blue has been stated to be of value by one or two writers, while the anti-malarial remedies given in India and elsewhere consist largely of diaphoretics in addition to quinine. Baths are not infrequently of considerable advantage. Finally, it should be again stated that no remedy has such a remarkable effect on the malarial organism as quinine; it seems to have a peculiarly toxic action on the parasites, and while it can hardly be stated that, if sufficient quinine is administered, the patient will for ever after be free from malaria, the most remarkable benefit is derived from its careful and painstaking administration.



XL. SLEEPING SICKNESS.

is the result of the presence of a parasite in the blood and the trypanosome. There are different varieties of this parasite, some of which may produce symptoms in animals, and when man is affected a form of lethargy called sleeping sickness may develop. The tsetse fly conveys the parasite from one animal to another, and the death-rate in humans and cattle, due, it was supposed, to the tsetse fly, is in many cases the result of the trypanosome. In certain districts of Africa a large percentage of the population have trypanosomes in their blood, and therefore there may be no symptoms produced by the parasite; but in other cases sleeping sickness develops, and when it is remembered that the period of incubation is at all events the period during which the trypanosomes produce no symptoms, may extend for a matter of years, it is not surprising to understand the difficulties which surround the disease. Sleeping sickness begins with the gradual development of weakness, associated with difficulty in walking and speaking, tremor in the limbs, and increasing pyrexia. The fever increases and the patient may die in a few months. Trypanosomes are found in the cerebro-spinal fluid as well as in the blood.

The **Prognosis** is grave and the **Treatment** at present is unsatisfactory. It has been found that certain aniline dyes exert an action on the organism somewhat similar to that of quinine on the malarial plasmodium. Arsenic has also been administered in a few cases.

DISEASES OFTEN GROUPED AS CONSTITUTIONAL DISEASES

I. MUSCULAR RHEUMATISM

(MYALGIA)

A PAINFUL affection of certain voluntary muscles, tendons, and fibrous tissues.

Etiology.—It is generally the result of rheumatism, and is associated with cold and wet, although in gouty persons there is also a predisposition to this affection. In some cases of acute rheumatism the muscles may suffer as well as the joints.

Clinical Features.—Pain in the back muscles is termed *lumbago*, in the intercostal muscles *pleurodynia*, and in the neck muscles *torticollis*, or stiff neck. *Lumbago* is very common after exposure to cold or wet when one is overheated. It fixes the patient's body as if in a vice, and causes the utmost agony upon any movement of the affected muscles. *Pleurodynia* is a similar involvement of the intercostal, pectoral, and serratus magnus muscles, a deep breath causing pain, or any muscular movement, such as in coughing. *Torticollis*, or stiff neck, is an involvement of the muscles at the side of the neck, and specially the sterno-mastoid. Pains in other muscles have received different names, but the term *myalgia* can be used in all cases. The affected muscles are tender to the touch, and are often definitely swollen: they may be particularly painful when the patient becomes warm in bed, and a certain amount of pyrexia is not uncommon.

Diagnosis.—The pain on muscular movement distinguishes this condition from *neuralgia*, while digital pressure is not

al in *pleurisy*, with which pleurodynia may readily be sed.

Prognosis.—In most cases prompt treatment is followed quickly rapid benefit, although there may be a tendency recurrence of the myalgia.

Treatment.—Give one of the salicyl group of remedies with an alkali, and remember that the prevention of movement of the affected muscles is the best means of securing comfort for the sufferer. Counter-irritation is sometimes useful, such liniments as liniment of turpentine being very efficacious, and blisters may also be used. Soothing applications, for instance the A. B. C. liniment, frequently give relief to the patient, and in certain cases acupuncture is followed by improvement, the needles being left in position for 5 or 10 minutes. A hot bath, hot fomentations, gentle massage, when necessary, the use of opium or morphia, are all methods of treatment.

II. CHRONIC RHEUMATISM

Definition.—There are obviously two distinct types of rheumatism included under this title, the first being merely cases of acute rheumatism, only less marked pain, less acute joint involvement, and less disturbance. But there are also many cases included under the term chronic rheumatism which are not the result of a specific organism, and with our present uncertainty of the pathology of rheumatic fever it is obviously not to be dogmatic. In this latter group of cases, muscles, fibrous tissue, and not uncommonly the fibrous tissue of the joints, are the site of the disease, and less commonly the

Chronic rheumatism is more common in persons who have been ill or are past middle life, and who are exposed to cold and dampness. It is closely associated with a rheumatic family history and varies greatly in severity according to the weather. Hard manual labour, and insufficient food are also factors.

Pathological Anatomy.—In the articular form there is great thickening of the capsule and ligaments of the

joint, with a limited amount of effusion, and the joints which specially suffer are generally the larger ones, and less frequently the small joints of the hands and feet. The muscles in the neighbourhood of a severely affected joint sometimes waste, and very often the patient suffers from permanent ill-health. Cases in which special nerves are involved—as, for example, the sciatic—will be referred to under diseases of the nervous system.

Clinical Features.—The pain is worst at night, when the patient gets warm in bed; it is more severe in cold, damp weather, is often accompanied by creaking in the joint or joints, and becomes easier after exercise or rubbing. The swollen joints are generally tender on pressure.

More or less malaise may be associated with exacerbations, and considerable anæmia and debility result, and not infrequently deformity of the joint. There is rarely much temperature, but there is a considerable tendency for the heart to be involved, specially in articular cases.

Diagnosis.—It is difficult to distinguish this condition from *rheumatoid arthritis*, under which disease reference will be made to the differential diagnosis, but in *gonorrhæal rheumatism* the history of a urethral discharge, associated with the long-standing and persistent involvement of often a single large joint, generally renders the diagnosis easy.

Prognosis.—Chronic rheumatism is apt to become a fairly constant companion when once it has persistently attacked one or more joints. It recurs with any wet or cold weather, but does not, as a rule, shorten the life of the sufferer.

Treatment.—It is wise in every case to try the salicyl group of remedies, and especially when renewed pain and swelling bring the patient to seek advice. Potassium iodide in 10 grain doses, and guaiac resin in 5-10 grain doses, in capsules, are often, however, more efficacious than the salicyl compounds. Counter-irritation, especially with iodine, fly-blisters, or the button cautery, is of the greatest value, while baths of all kinds, and especially radiant-heat, electric, and alkaline baths, are beneficial; in fact, any hot-water baths associated with massage and passive movement of the affected joints often yield satisfactory results. Where possible, recommend a warm, dry climate if the patient can afford to escape the severities of the home winter. Dress warmly, and study the

7, remembering that in starved patients good feeding is
ie, while in over-fed sufferers from this disease restriction
; may be necessary.

III. RHEUMATOID ARTHRITIS

(ARTHRITIS DEFORMANS. OSTEOID ARTHRITIS)

ONIC affection of various joints, characterised by definite
s in the cartilages and synovial membranes, and gener-
llowed by new bone formation, and always by marked
ermanent deformity. The evidence that this condition
riated with a specific organism appears to be convincing.
iology.—Most patients suffer between the ages of 30
, and as a rule the more acute cases occur in the younger
. It is a disease which is certainly more frequent in
and appears in them to be the result of any excessive
such as repeated pregnancies or protracted lactation.
one joint alone is involved, and where the condition is
ronic, a slightly larger proportion of men suffer than

hereditary history is generally one of joint affection,
es of gout, and sometimes phthisis.

al injury seems to predispose to the mono-articular
t cold, worry, fatigue of mind and body, and insuffi-
d are amongst the most common etiological factors in
articular variety.

all bacillus has been described by Ballantyne, Bloxall,
rs, as occurring in the synovial fluid of affected joints,
rimental inoculation with a culture of the bacillus
ed results in animals closely corresponding to those in
ough it is difficult to believe that in all cases a
origin can be proved. It should be remembered that

gonorrhœal history has been obtained in a consider-
ber of patients affected with this disease, and it is
hat in certain cases the gonococcus may really be the
ganism.

nber of authorities refer to what may be called the
eory in attempting to explain the causation of the
id there is at first sight a similarity between the

Charcot's joint affection of locomotor ataxia and rheumatoid arthritis, but the statement that the wasting of muscles in the neighbourhood of the affected joints is the result of a neuritis which is a definite etiological factor in the disease, is improbable. It is true that lesions of the nervous system, such as occur in infantile paralysis, cause atrophic changes in muscles, bones, and skin, but only to a limited degree are joints affected.

Pathological Anatomy.—The first change is probably in the cartilages of the affected joint, the cells of which proliferate, and fibrillation occurs at right angles to the articular surface of the joint. Soon afterwards the softened cartilage is rubbed away, and when the bony surfaces come in contact they become eburnated, and a considerable amount of creaking is produced in the joint on movement. Associated with this change is an increased vascularity and a coexistent development of fresh cartilage at the periphery of the joint. The synovial fluid is increased in amount, and at an early period there is deposition of bone in the newly formed cartilage, causing "lipping" and greatly diminishing free movement. In the periosteum also fresh bone may form. This development of new bone in connection with cartilage and periosteum is called *osteophytic*. It can be easily understood how repeated attacks of inflammation, with increase of fluid and change in the cartilages, will in time cause marked disorganisation of the joint, while ankylosis is by no means uncommon.

In the **mono-articular form** of the disease there is apt to be absorption of bone, the existing cancelli being in part removed by osteoclasts.

In the form of the disease described as **Heberden's nodes**, small osteophytic outgrowths develop at the bases of the middle and terminal phalanges, usually lateral in position. When present they very frequently appear to limit the advance of the disease towards other joints.

In many cases there is muscular wasting in the neighbourhood of affected joints, and sometimes spasm of atrophied muscles may add to the deformities brought about in the joint cavities; hyperextension of the distal interphalangeal joints is not uncommon.

Clinical Features.—**Type 1.**—**The Polyarticular Form** may be acute, subacute, or chronic, and in the more acute

is specially common in young persons, in whom it may be associated with glandular enlargement. It commences in the hand, often one of the small joints of the hand, and rapidly spreads; it is generally symmetrical, and the joints are painful, and the hands have a typical bulbous-like appearance with a considerable amount of local hyperæmia. Repeated attacks may occur, one subsiding, but usually leaving definite joint changes, and with a number of attacks much deformity inevitably results.

Pain in the ball of the thumb has been described as an early symptom of the disease, and fresh attacks commonly result from any exposure to cold or any physical exertion.

There are four typical features which are often associated with the forms included in this type:—

Pigmentation over or near the affected joints, sometimes in other positions, is extremely common, and may even be an early and suggestive feature of the disease.

Sweating, especially in the palms of the hands and the feet, is frequently present, and is more severe during attacks.

A definite, fine tremor may be seen in the hands and fingers.

There is often an exaggeration of the tendon reflexes. There is sometimes tachycardia, and neuralgic pain is common in the ball of the thumb and the ulnar side of the wrist.

In a certain proportion of patients the temporo-maxillary joints are affected, and occasionally the intervertebral joints, especially in the region of the neck.

Many patients suffer for years from the disease, but, eventually, in not a few cases a time arrives when the disease is arrested and there is immunity from renewed attacks. The twisting of joints is in the direction of flexion, with deviation of the terminal phalanges of the fingers, which are extended, and the wrists are bent towards the ulnar side.

Still has described this type of rheumatoid arthritis in children, and associated it with remarkable enlargement of lymph glands and spleen.

2.—Mono-articular Form.—This form is common in adults, and affects the knee, hip, shoulder, and more rarely the intervertebral joints. Shortening of bone may be

noted, and it tends to follow local injury. The general symptoms, such as pigmentation, local sweating, tremor, etc., are not present.

Type 3.—In this type **Heberden's nodes** develop. As already noted, they form at the bases of the terminal phalanges of the fingers, and are cartilaginous or osteophytic outgrowths from the joint cartilage. They are associated with a limited amount of swelling, pain, and the usual changes in the cartilages described under Type 1. The disease is usually limited to the smaller joints just mentioned, and generally becomes arrested at an early period. Heberden's nodes are said to be more common in women.

Diagnosis.—*Gout, chronic rheumatism, and gonorrhœal rheumatism* may all be confused with this disease. In acute gout the usual joint first, and often alone, affected is the metatarso-phalangeal joint of the great toe, and the definite history of the attacks, with generally a hereditary gouty tendency, should render the diagnosis easy. In chronic cases of either disease there is much more difficulty, although a careful study of the diet, of the urine, and of the history of the joint attacks is usually very distinctive in a case of chronic gout.

In rheumatism relief is generally obtained with salicylate of soda, whereas in rheumatoid arthritis that drug is generally valueless.

Gonorrhœal rheumatism certainly resembles Types 1 and 2 of this disease, but the history of the case, if obtained, should prevent any mistake, and pigmentation, sweating, and tremor are not present except in cases of rheumatoid arthritis (Type 1).

It should be mentioned that the temporo-maxillary joint may be affected in gonorrhœal rheumatism and in rheumatoid arthritis, while it is extremely uncommon to find it involved in gout or chronic rheumatism.

Prognosis.—At the best, a progressive case necessitates long and often energetic treatment before it is even safe to say that the disease is arrested. In the 2nd and 3rd Types, and especially the 3rd, the condition is limited, and the prognosis is more favourable.

Treatment.—The etiological factors must be carefully considered, an insufficiently fed patient should be ordered a nourishing and easily digested dietary, and in no disease is the

the use of port wine and malt liquors more satisfactory. It must be carefully guarded against, and if possible the patient should seek a dry and warm climate.

Internal treatment by drugs is hardly so successful. Cod-liver oil, malt extract, and similar agents are good, while iron and arsenic are often advantageous. Potassium iodide and salicylate of guaiacol have been strongly recommended.

Local treatment is more important; persistent painting of affected joints with iodine is of very great value, while some benefit may result from the use of hot-air baths, electric hot-water baths of all kinds, and the high-frequency currents. Douching and wet packs are amongst the numerous hydropathic methods of treatment which suit different

forms of treatment should be combined with massage, and the massage should include movements which tend to increase the usefulness of the affected joints. In some cases removal of an ankylosed joint has been performed in order to render the limb more serviceable.

IV. GOUT

(PODAGRA)

Gout is a disease, or one due to a form of faulty chemistry, the nature of which is as yet not clearly defined. It is associated with an excess of uric acid, possibly formed in the body as a result of the affection, and with the deposition of urate of soda in joints, accompanied by typical attacks of arthritic inflammation. Excess of uric acid does not necessarily imply excessive consumption; it is more probably due to defective excretion. The disease may be (1) Acute or (2) Chronic, and there are several forms of gout to which the terms (3) Suppressed and (4) Tophaceous are given.

Etymology.—There is a strong hereditary predisposition, and in gouty families the dangerous period is reached when middle life is passed. Probably in fully one-half of the cases there is a definite hereditary history of gout, although some generations are missed over and the legacy handed on to succeeding children. The male line is credited with the stronger

hereditary taint, and males suffer more markedly than females. It is most common between the ages of 30 and 50, but it may occur at any time of life.

An excessive amount of alcohol is certainly responsible for many cases of the disease, and especially the sweet, heavy ales affected by English palates as distinct from Scottish.

Overeating is another etiological factor in not a few cases, and is apt to lead to gout if it is associated with a sedentary life and too little exercise. Lead poisoning is responsible for the production of some of the more chronic cases of gout, and gout is peculiarly common in persons whose work necessitates the use of lead.

Notwithstanding these fairly definite etiological factors, the pathology of gout is distinctly mysterious. Garrod has suggested that there is a lessened alkalinity of the blood, and that, as the result of diminished elimination, an excess of uric acid occurs. He asserts that urate of soda is not held in solution, and is suddenly deposited during the gouty paroxysm in the joints. Haig considers that the blood is less alkaline, and therefore less able to keep the uric acid in solution. There is no excessive manufacture of uric acid. Roberts considers that uric acid circulates as a quadriurate, which is freely soluble and easily eliminated by the kidney. If it lingers in the blood and should happen to come across sodium carbonate, it takes up an additional atom of base, becoming as a result a biurate, which is much less soluble, and readily crystallises out in parts where the circulation is less rapid, and especially in the presence of sodium chloride, which is found in the lymph in the synovial sacs. Levison agrees with this statement, but adds that uric acid is obtained from the breaking up of white blood corpuscles, and therefore is proportionate to the leucocytosis present. Ord believes that there is a general or local disintegration which precedes the deposition of the urate of soda in the joint, and Ebstein considers that this joint process is a form of necrosis. Sir Dyce Duckworth holds that there is a peculiarity as regards the innervation of hereditarily gouty individuals, which influences the production and the elimination of uric acid.

It seems probable that gout is due to faulty excretory processes, and that the kidneys may to some extent be held to be responsible.

Pathological Anatomy.—There is a deposit of urate of in the cartilages of the affected joints in the form of ar crystals, and these are also to be seen in the ligaments, the bursæ, the tendon sheaths, in the skin over the joints, and the cartilages of the ears, nose, and elsewhere. In the joints yellowish-white lines are seen, indicating the deposit of crystals. There is apparently an excess of uric acid in the blood, although this is not peculiar to gout alone, and Rayer's experiment of the thread in blood-serum is not always reliable, even in cases of acute gout. It consists in obtaining a small amount of blood-serum from the patient, adding 2 or 3 drops of glacial acetic acid, and placing a thread in the mixture for some 30 or 40 hours, when uric acid crystals may be found on the thread.

unquestionably the metatarso-phalangeal joints of the big toe are the most frequently affected, although any joint may be affected.

Arterio-sclerosis, with hypertrophy of the left ventricle, sometimes primary cirrhosis of the kidney, form a not infrequent combination in gouty patients, and more especially in cases of chronic gout.

General Features.—(1) **Acute Gout.**—There are nearly always premonitory phenomena, the patient feeling out of spirits, restless, dyspeptic, or peculiarly irritable. The acute attack begins with pain in, and tense shiny swelling of, the metatarso-phalangeal joint of the great toe, or more rarely of another joint. The veins in the skin become distended, and the joint throbs much like a suppurative arthritis. There is marked dyspepsia, concentrated, highly coloured uratic sediment, and the attack comes on during the night or towards morning. The pain is excruciating, and the affected joint swells rapidly; the temperature may reach 102° F. or higher, but towards morning the attack wears off. A return attack is probable during the next night, and for four or five consecutive nights there may be a considerable amount of swelling. As the attack passes off, the pain is less and more diffuse, but attacks are apt to recur from any trivial error in diet, and in severe cases at shorter and shorter intervals. In the attacks both the uric acid and phosphoric acid in the blood fall to a very low level, rising, however, as the disease develops. Frequently the patient feels much better after the attack, although repeated attacks of acute gout pull

down the strength and render the patient extremely miserable. The skin over the affected joint may desquamate.

(2) **Chronic Gout.**—In chronic gout the attacks are longer and more joints are affected. Deposition of urates occurs in the cartilages and ligaments around the joints, so that the joints become swollen and eventually much deformed, and osteophytic deposits are very common in connection with them. The feet and hands, sometimes the elbows and knees, suffer, while gastric disturbance is extremely common, and not infrequently arterio-sclerosis is present. It is in chronic gout that the uratic deposits in the skin called *tophi* are best seen—the so-called chalk-stones with which, if they occur in the knuckles, the patient can write upon a slate. There is not infrequently a trace of albumin in the urine, and from time to time attacks of joint pain occur. There are many complications of gout, to which reference will be made under the heading of Irregular Gout.

(3) **Suppressed Gout** implies serious gastric, intestinal, cardiac, or other disturbance, coincident with the disappearance of pain in the affected joints. Vomiting and pain are the most frequent gastric, and diarrhoea the most frequent intestinal symptoms, while dyspnoea with irregular action of the heart may occur as cardiac manifestations. In rare cases delirium, and sometimes even apoplexy, may be simulated in suppressed gout.

(4) **Irregular Gout.**—In all gouty families there is a tendency for certain gouty manifestations to appear, and many of these are in reality the complications which may occur in any gouty patient. They include in the *Alimentary System* a tendency to gastric attacks of biliousness, in the *Circulatory System* palpitation, irregular action of the heart, and not infrequently the development of arterio-sclerosis. In the *Respiratory System* bronchitis is very common; in the *Integumentary System* gouty eczemas and acne rosacea are peculiarly prevalent. In the *Urinary System* uric acid calculi are not infrequent, and associated with them or with the passage of gravel, urethritis may develop. In the *Nervous System* headache and especially neuralgia may annoy the patient. Apoplexy, probably as the result of the arterio-sclerosis, is not uncommon, and eye affections should not be forgotten. There may be inflammation, such as iritis or

; glaucoma may occur, but is probably accidental. frequently the patient suffers from attacks of episcleral inflammation.

Many of the conditions mentioned under the different forms of gout, and especially in connection with irregular gout, might be more truly termed complications rather than features of gout itself, and we are probably justified in including inflammations of serous membranes, such as of the pericardial sac, peritoneum, and meninges, under the head of complications; together with uræmia they are apt to be associated with the final period of life of our patients.

Prognosis.—Much stress should be laid on the family history, and also on the patient's habits as regards food and drink.

Where tophi are present the typical acicular crystals of soda may be seen under the microscope, if "chalk-pur" can be obtained and crushed. There is certainly an increase of uric acid in the blood, although the exact significance of the increase in the urine during an attack is by no means demonstrated. The gouty attack is very typical, with the exception of chronic cases, in which the clinical features are less definite. When it is remembered that irregular gout is almost invariably a hereditary legacy, the most obscure clinical features may acquire their due significance when diagnosed as gout.

Prognosis.—Acute gout is readily recovered from, but there is a tendency to recurrence. Chronic gout leaves permanent changes in the joints, and irregular gout, notwithstanding frequent manifestations, is generally amenable to treatment. Suppressed gout is assuredly the most dangerous, and the sudden onset of severe gastro-intestinal disease, serious cardiac disease, or other manifestation of the paroxysmal pains of an attack of gout, should lead the physician careful in giving a too favourable prognosis.

Treatment.—(1) *Treatment of the Attack.*—The affected limb should be elevated, and, if the patient is confined to bed, the limb should be placed on a pillow; the joint or joints should be wrapped up in cotton wool, and fomentations, especially with opium, will be found very soothing. Order a powerful purgative, probably a dose of calomel or blue pill, followed by a saline draught in the morning. In most

cases, provided the heart is satisfactory, a dose of tincture of colchicum seeds (m 15-20) should be at once administered, and may be repeated twice or even thrice in the 24 hours; the patient may be encouraged to drink freely of lithia water, or citrate of lithia may be given in 10 - 20 grain doses thrice daily. The diet should be of the simplest possible description, by preference milk diet alone, or plain soups, and on no account should sugar in any form be eaten. It is better to recommend the patient to use saccharin or saxin in place of sugar, both in cookery and also for sweetening tea or coffee. An excess of starch must also be strictly prohibited. The patient may be permitted to have fresh fruit or vegetables, but oranges and fruits that are not too sweet should be preferred.

Where they can be obtained easily, hot-air or radiant-heat baths, applied locally to the affected limb, prove very efficacious, and once the affection has yielded and become less acute, a course of baths are advantageous; these are referred to later.

(2) *General Treatment of Gouty Patients.*—Persons liable to attacks of gout should be extremely careful with regard to diet. Their meals should be in strict moderation, care being taken to eliminate sugar, pastry, sweet fruits, especially preserved fruits, while both starchy articles and butcher meat must have a carefully balanced proportion in the patient's ordinary daily diet. Fish and white meat are good, but beef, pork, and salt meat should be sparingly eaten. Foods rich in nuclein, such as sweetbreads, and meat extracts containing excess of xanthin bodies, should be avoided. Potatoes, artichokes, and starchy vegetables are better eschewed, while cabbage, spinach, cauliflower, and tomatoes can be eaten freely.

A very important point in treatment is unquestionably the matter of liquor. A small quantity of whisky or spirit without sugar may be ordered when necessary. Certain Moselle wines, such as Berncastler Doctor, are free from sugar, and may be used by the patient, while cider of the dry variety has been recently recommended, and bitter beer, although assuredly undesirable, is not so hurtful as sweet ales or porter. For wealthy patients dry champagne may be allowed in moderation, but port wine, burgundy, and all heavy, sweet

should be strictly prohibited. Roberts suggests that sodium chloride should be used in place of common salt at times, only the taste is certainly far from agreeable. He recommends the free use of fatty articles of food, such as butter. Most gouty patients benefit from alkaline mineral waters, although it is possible that the benefit is derived more from the flushing of the system than from any inherent the waters may contain. It is true that in the treatment of gout, important factors are the careful management at home and abroad, where special springs are used, freedom from home and business worries, and a healthy diet with plenty of exercise. Many baths have, however, obtained a highly good and well-merited reputation, such as Bath, Tunbridge, and Harrogate in this country, and Carlsbad, Aix-les-Bains, Contrexéville, Homburg, and many others abroad. For a few patients the mere use of a daily purgative saline is a good treatment, and it is probably better to substitute magnesium or potash salts for soda. Massage is useful where the joints have become more or less affected, and careful dieting, together with the prescribed amount of exercise, is very helpful.

The clothing should be warm, but not excessive; probably flannel or wool should be worn next the skin. It is wise, if possible, to send gouty patients, who are suffering from the influence of our climate, to a suitable health resort abroad, as this is helpful in not a few cases.

In chronic gout, potassium iodide, guaiacum, and colchicum may be administered, while iron and arsenic are of advantage if the patient is anæmic.

Attention has been made of so-called uric acid solvents. Sodium bicarbonate (gr. 15-30 daily) has a considerable reputation, but its effect is disappointing.

V. RICKETS

(RACHITIS)

Rickets is a disease of infancy and early childhood, generally commencing before the end of the second year, and characterised by excessive development of cartilage, especially in con-

nection with the epiphyses, and by delayed and defective ossification of many of the bones of the body.

Etiology.—It is essentially the result of a dietetic error. It occurs where children are bottle-fed, and where the milk used is “condensed” or skimmed milk and poor in fat. There are, however, many children who are breast-fed, or fed upon fresh milk, but who in addition are given unsuitable articles of diet, such as tea, or part of “anything that is going,” and these children are specially prone to develop rachitic tendencies if they are also placed in the midst of insanitary surroundings.

In the homes of the rich, a child may develop rickets who is being fed upon predigested food, and it is worth remembering that special foods of the starchy order are liable to produce a combination of scurvy and rickets. The excessive use of carbohydrates in the dietary may be the important factor in certain cases, but it should not be forgotten that the slightest dietetic error takes greater effect on delicate children and those who are not in hygienic homes.

Pathological Anatomy.—There is a great increase of cartilage between the shaft and epiphyses of the long bones, and it is abnormally vascular. Ossification is therefore delayed and is imperfect. The bones which show the earliest changes are the ribs, especially next the sternum, the lower end of the radius, and the bones of the skull. These and other affected bones are found to be unduly soft, being easily cut with a knife, and on section, especially near the epiphyses of the long bones, there is undue vascularity with irregular islands of ossification. The bones yield to pressure, and the typical changes seen in after-life are the result of the consequent twisting and bending. The periosteum is also unduly vascular, and probably this vascularity causes great delay in ossification in its neighbourhood. There is removal of bone already formed, and the medullary canal is found to be abnormally enlarged.

Clinical Features. — The disease advances insidiously with marked debility, malnutrition, and nocturnal restlessness. The child sweats freely at night, and especially over the head, while dandling the child causes great pain. The little patient becomes emaciated, and soon the enlargement of the epiphyseal ends of the long bones is manifest. The delay

ossification causes important changes in the shape of the t and the development of the body generally, while dentition so protracted.

The "rosary" ribs are extremely typical, and consist in a bead-like enlargement corresponding to the junction of cartilage and bone. There is indrawing of the costal cartilages and the sternal ends of the ribs, tending to produce a keel-shaped sternum, while Harrison's sulcus, so commonly seen in rachitic chests with which rickets is not infrequently associated, is due to indrawing of the chest wall above the insertion of the diaphragm. The lower ends of the radius and ulna and the tibia become enlarged, and contrast markedly with the thin, poorly formed shafts of the bones. Should the child endeavour to walk, the typical bow-legged condition results; if the child creeps about on the floor, the tibiae may splay outwards and the feet slightly everted, because it is as if the legs behind it much like the hind fins of a sea-lion. The genu valgum, or curvature of the neck of the femur downwards and outwards, giving to the patient a peculiar waddling gait, is an occasional result of this disease. The arm bones may also be affected. Should the child use the arms to assist in progression, the pelvis undergoes marked alteration, forming the typical rachitic or flat pelvis which causes such trouble in labour. The bones of the skull remain long unossified, and there is a considerable degree of hydrocephalus associated with it, but in any case the closure of the fontanelles is delayed. A rachitic head is square-shaped, and the frontal bosses are unduly prominent, giving the patient the appearance, at least, of great mental power. Sometimes cranio-tabes is present, by which is meant yielding of the bones of the skull to digital pressure, and is due to defective ossification. Rickets is also responsible for knock-knee, flat-foot, and other deformities, but these results of the disease occur usually at a much later period than the phenomena just mentioned. Spinal curvature is often present, although the degree is not so great as in cases of tubercular caries. It is due partly on the softness of the bones and partly on the weakness of ligaments and muscles. In later life, when ossification is completed, the malformations become permanent.

While these changes are developing the child suffers in

other ways. Bronchitis is extremely common and may be very severe, the soft, easily moulded chest wall readily yielding to the pressure of coughing. Most rachitic children are also anæmic, although the degree of anæmia in a simple case is not generally very extreme. Amongst the complications of rickets may be mentioned laryngismus stridulus, convulsions often associated with dentition, and tetany. As a rule the liver and spleen are enlarged in rickets. Teething is invariably delayed, often the first tooth only appearing after a year or fifteen months, and the teeth are apt to be irregular and have an early tendency to become carious.

There is a variety of rickets occurring at a later period than usual to which the term late rickets has been applied. It appears in children of 3 or 4 years old, sometimes even at a later period, and is associated with epiphyseal enlargements of bone, muscular weakness, and marked sweating. Generally various deformities result, such as flat-foot, knock-knee, or spinal curvature.

Diagnosis.—It is most important in children to keep before the mind the possibility of *scurvy-rickets*, because there is no other disease with which it is possible to confuse this condition. Bronchitis, anæmia, and many other permanent or temporary affections may depend to a certain extent on pre-existing rickets, and the rachitic element in these cases should not be overlooked.

Prognosis.—Death may occur as the result of some inter-current disease, but in many cases the child survives, with more or less permanent deformity, and the deformity in question may even endanger life, as for example the flat or rachitic pelvis during labour.

Treatment.—The great causal factor in rickets indicates the cure. The child should be at once placed on suitable diet, and that means milk with plenty of cream, and he should at the same time have abundance of fresh air and sunshine, and should be clad warmly, although not excessively. In older children a proper proportion of nitrogenous and carbohydrate ingredients in the diet should be ordered. There are certain drugs which may be given in addition; these include cod-liver oil, syrup of the iodide of iron, the syrups of the phosphates and hypophosphites, and arsenic in cases in which anæmia is very severe. Laryngismus stridulus must be treated on the lines suggested

describing that disease. All that need be said here is antispasmodics, such as chloral and bromides, are of great fit.

Care should be taken to prevent increasing deformity, where possible to straighten twisted limbs. Sometimes casts can be intermittently used for this purpose; in other cases wedges of bone may have to be removed. Lastly, no child is more benefited by sea air than rickets, and if possible it is wise to have children so affected resident at the seaside, and as much out in the open air as the weather will permit.

VI. DIABETES MELLITUS

persistent excretion of sugar in the urine with definite deterioration of health. It is a matter of opinion whether treatment should be applied to cases in which the passage of sugar is more or less temporary. It seems preferable to treat such temporary excretion of sugar, glycosuria.

The carbohydrates of the food are stored up as glycogen in the liver, and it is difficult to ascertain what part the liver plays in the production of glycosuria. It seems certain that the normal secretion of the pancreas aids the tissues in some way in the destruction of sugar, thus preventing its excretion by the kidneys. The injection of large quantities of sugar into the blood has not resulted in the production of glycosuria, and removal of the liver, experimentally, causes the amount of sugar normally present in the blood to disappear. In diabetes mellitus there may be possibly (1) an over-production of sugar by the liver, or (2) a diminished destruction by the tissues, and in both of these supposed conditions the influence of the pancreatic secretion may be the factor concerned. It is conceivable that the sugar brought to the liver by the portal vein is not converted into glycogen, or that there may be excessive conversion of glycogen into sugar (Hamilton). According to Seegen and von Noorden, if an excess of sugar is present in the tissues, some of it will remain in the blood.

Glycosuria of a *temporary* nature may be present as the result of the following conditions:—An excess of sugar in the blood will cause a temporary glycosuria to appear, the

amount needed depending much on the personal equation of the individual, and also on the existence of any disease from which he may be suffering. Sugar is also present in obesity, especially in oldish persons, in chronic alcoholism, in exophthalmic goitre, and sometimes in certain of the continued fevers. The administration of phloridzin, opium, chloral, and other agents, especially in certain individuals, and chloroform and ether anæsthesia, may produce a temporary appearance of sugar in the urine. It must, however, be remembered that if the only test used for detecting sugar is copper reduction, there are other agents besides glucose also capable of reducing the copper salt.

Etiology.—No disease presents such difficulty as regards an accurate etiology and pathology. There is sometimes a hereditary predisposition, and the disease is certainly commoner in males. Mental strain, gout, syphilis, and excesses—mental, moral, physical, or alcoholic—may all cause or predispose to the disease. Injury or disease of the brain or spinal cord, and especially in the region of the 4th ventricle, has resulted in diabetes, and quite a number of cases are definitely associated with atrophy or interference with the secretion of the pancreas. It has resulted from, or at least followed, an acute illness such as pneumonia or a continued fever, while in certain instances it is said to have ensued upon drinking cold water when overheated. Unfortunately in many cases there is no reliable history whatsoever.

It should be remembered that in stout persons, in those who are gouty, and especially after middle life, there is a tendency to a temporary or in other cases a permanent, but slight, degree of glycosuria.

Diabetes mellitus is not common, but it is increasing, and in Paris, for example, the death-rate from the disease is making rapid strides. It claims, proportionately, most victims among Jews, but the fact that glycosuria and diabetes mellitus are frequently considered synonymous, renders many series of statistics of little real value.

Pathological Anatomy.—There is no question that a tumour, cyst, or softening at or near the “sugar puncture” of Claude Bernard in the floor of the 4th ventricle may cause diabetes, and also that atrophy of the pancreas or the presence of a pancreatic cyst or tumour may act in the same way.

There are, however, many cases of diabetes mellitus in which the pathological anatomy is eminently unsatisfactory, most of the conditions to be described are either the results of the disease, or may be met with after death in many totally dissimilar affections. Thickening of the membranes of the brain; enlargement of perivascular spaces; tendency to colloid masses and corpora amylacea in brain and degeneration, especially of the posterior columns of the spinal cord, multiple neuritis, and changes, which can hardly be called normal, in the cells of the semilunar ganglia, the sympathetic ganglia in the neck, and elsewhere, have all been described in the literature of this system. In the heart there may be fatty degeneration, and in the blood in a very few cases there is a great excess of sugar, there is generally an increase in sugar, and a marked increase in alkalinity, possibly due to the formation of lactic and other acids, while glycogen occurs free in the erythrocytes of the blood and in many organs of the body.

Many patients suffering from diabetes mellitus acquire emphysema, and may die of phthisis pulmonalis, while pneumonia of the lobar type sometimes develops, and gangrene of the lung, secondary to either phthisical or pneumonic infection, is by no means an uncommon mode of fatal termination. The liver, which we should expect from many cases in the past to show some definite and diagnostic changes, may reveal nothing beyond evidences of glycogen in the cells and fatty degeneration, while the kidneys usually show merely a glycogenic infiltration.

Physical Features.—When diabetes mellitus occurs in children the disease assumes a very acute form, whereas in adult patients it is more chronic. The first symptoms are increased thirst and polyuria, and there is rapid loss of weight with progressive weakness. In certain cases a history of apparently trivial glycosuria precedes the disease. The emaciation associated with the dry skin, the scanty hair, and the sallow expression of face, are very typical.

Digestive System.—The tongue is red and sometimes dry, the mouth dry, the teeth bad, and often the breath has a peculiar odour of acetone, and the gums may be swollen and inflamed. There may be marked dyspepsia with acid eructations, or the appetite may be large and digestion good. Constipation is almost always marked.

Hæmopoietic System.—There is an excess of sugar in the blood, the normal 1 to 1·7 per 1000 being often increased to 2·7 up to 5·7 per 1000. Reference has already been made to the rare cases in which fat appears in the blood. The alkalinity is also reduced, probably as the result of the presence of diacetic or other acid. There is frequently anæmia with reduction of red cells and hæmoglobin.

Circulatory System.—There is usually no marked change in the heart or pulse, unless a secondary nephritis has been set up or the patient has developed diabetic coma. In the former condition cardio-vascular changes may develop, and in the latter the pulse becomes excessively feeble.

Respiratory System.—With the exception of the complications of phthisis and pneumonia, to which many diabetic patients fall victims, there is only the remarkable dyspnœa to mention, which, however, is more appropriately described under diabetic coma.

Integumentary System.—The dry skin has already been referred to, and, probably as a result of the irritating qualities of sugar in the sweat, there is great liability to acne spots, boils, and carbuncles, perhaps more specially in the older diabetic patients. Eczema, due to the sugar in the urine, gives rise to much irritation and discomfort about the genitals, and a peculiar bronzing of the skin has been recently attributed to diabetes. There are other rare diseases of the integumentary system, such as xanthoma, but it is hardly necessary to refer to these here.

Urinary System.—The urine increases from the normal 50 oz. to 200 or 300 oz. in the day. The colour is typically greenish-yellow; the specific gravity is almost always high (1030-1050). A low specific gravity may be present in milder cases, and too much importance should not be attributed to the daily reading of the urinometer. The amount of sugar present varies greatly, and may rise as high as 10 per cent, the total amount of sugar being 10-25 ounces. It has been found that the urea, which is also invariably increased, bears a direct proportion to the amount of sugar, in most cases being about 1 part to 22 of sugar, and the total amount of urea secreted may reach 1000 or 2000 grains per day. Phosphates are also in excess in the urine. The waste of sugar is greatly increased by an ordinary diet, and muscular

se, and in fact any severe strain on the patient, usually the sugar excretion. Generally the urine passed during day contains more sugar than that passed during the night; the latter, in mild cases, may even be free from sugar. Sometimes, owing to the presence of the torulae, the urine becomes cloudy, fermentative changes taking place in the sugar. The urine often contains acetone, which gives a peculiar odour to it, while certain of the bodies which probably form acetone, such as diacetic acid and β -pyruvic acid, may also be present.

In connection with the estimation of the total amount of sugar in the urine it is well to remember that the fermentation method is the most accurate, and that, provided there is no alcohol present in the urine (which rotates the ray of light to the left), the polariscope also gives an extremely exact estimate of the percentage of sugar, owing to the fact that it rotates polarised light to the right.

Perhaps it is well again to remind the reader that there are other agents besides glucose which reduce copper solutions, among these should be mentioned glycuronic acid, pyronin, hydrochinon, and even uric acid.

Nervous System.—Practically all the phenomena related to in this system are complications, many of which, therefore, it is impossible to omit from our description of the

Diabetic Coma.—This was first described by Kussmaul, and is an extremely common cause of death in *young* diabetics, it now accounting for quite $\frac{9}{10}$ ths of the death-rate in these cases. The onset is often gradual and insidious, the urine falling in amount and the sugar becoming greatly lessened, but it should be remembered that it may ensue on too energetic limiting the patient to a non-diabetic diet, or may result from constipation. The pulse becomes quick and the surface of the body cold and clammy, the patient always complains of severe abdominal pain, followed or preceded by headache, and generally there is a considerable amount of excitement. One prominent feature is *air-hunger*, as given by Kussmaul to the extraordinary dyspnoea associated with diabetic coma. Without any obstruction to the passage of air, the patient seems totally unable to obtain sufficient oxygen.

It is difficult to say what the toxic agent present is, but probably β -oxybutyric acid in the blood is partly responsible. There are three other theories formulated to explain diabetic coma which should be mentioned: the *first*, that fat emboli occur in the lung; the *second*, that it is really due to uræmia, the result of a superadded Bright's disease; and the *third*, that it is an auto-intoxication from the bowel, and that constipation is largely responsible for precipitating an attack. Whatever the cause may be, unfortunately, in most cases, the patient sinks rapidly into coma unless treatment gives prompt relief.

(2) Peripheral Neuritis.—This develops in certain cases of diabetes, and it has been suggested that the neuralgic pains sometimes associated with the disease may have their origin in this way.

(3) Cord Affections.—It is a common feature of diabetes mellitus to observe that the knee-jerks are lost, and the wrist-jerks are usually also absent, while sometimes evidence of involvement of the cord, as in locomotor ataxia, shows itself.

With regard to special senses, cataract is of frequent occurrence. It is generally of the soft type, while sometimes various defects of vision, such as paralysis of accommodation or central scotoma, make their appearance in the course of the disease.

Diagnosis.—As a rule the diagnosis is easy. It is far more difficult to diagnose a slight degree of glycosuria where it may chance that glycuronic acid or other copper-reducing agent is present. But the amount of sugar found in the urine in diabetes is sufficiently great to prevent any possible mistake of this kind.

To distinguish diabetes mellitus from glycosuria, it is only necessary to remember that in diabetes mellitus it is not possible that diet alone can remove the sugar from the urine, while in most cases of glycosuria a careful dietary will cure the condition, and even where it does not, there are none of the grave symptoms and signs which characterise a typical diabetic case.

The colour-reaction of eosin and also of methylene blue on the red blood corpuscles has been used in diagnosing diabetic coma. With eosin they stain less deeply than do normal red cells, and methylene blue gives a yellowish-green colour which is distinctive.

Prognosis.—Under the age of 40 the prognosis is grave, after that age it is more favourable. Rapid and permanent relief from dietetic and other measures should also warrant a favourable prognosis. The appearance of nephritis, of pneumonia, or of pleurisy pulmonalis, and, it need hardly be added, the development of diabetic coma, are all of grave import, and, as already stated, diabetic coma is the chief cause of death in young patients. In patients under 40, even with the utmost care as to diet and over-exertion, bodily or mental, a case will not usually live for more than a few years, possibly only months, as in elderly persons the disease may rather be a permanent glycosuria than a true diabetes, and the patients may live for many years.

Treatment.—The first point of importance is to find out the amounts of urine, of sugar, and of urea excreted by the patient, and if possible on an ordinary diet. After a few days or a week we should endeavour, in the second place, to find the diminution in quantity which can be effected by a nitrogenous dietary. After another week on this diet, if it may be supposed that the average figures are fairly constant, we can attempt treatment by drugs. It may be again stated that the complete withdrawal of carbohydrates from the diet does not arrest the excretion of sugar in the urine in a severe case of diabetes, although it greatly diminishes the amount. It should not be forgotten that a rigid nitrogenous diet presents an element of danger, inasmuch as it is liable to have conduced to the development of diabetic coma in very severe cases, and therefore it is wise to allow a little of a potato, or some such carbohydrate ingredient.

In studying the question of dietary we must endeavour to provide the patient a diet under which he will not lose weight, and in which, as far as possible the sugar will not increase in amount. Williamson suggests that it is desirable to study the caloric producing qualities of the different articles of diet precisely, and this is expressed in calories (a calorie is the amount of heat required to raise 1 kilogramme of water 1° C.). One gramme of proteid and 1 gramme of carbohydrate each yields on oxidation about 4 calories, while 1 gramme of fat yields about 9 calories. The quantity of these three substances in the patient's diet is calculated, and their value expressed in calories. From the total the value of the sugar lost in the urine,

estimating by calories, and the remainder ought to be not less than 2300 calories daily. This is the value in calories of the food required daily by a healthy man not engaged in specially arduous physical work.

The following articles of diet may be permitted :—

Fish of all kinds, excepting those with large livers, which should be avoided, at least in excess; butcher meat of all kinds, excepting liver; poultry and game.

Soups, milk, butter, eggs, cheese, oils, and fats.

Green vegetables, tomatoes, radishes, asparagus, cucumbers, mushrooms, and most kinds of pickles (unsweetened).

The following substitutes for bread are more or less free from carbohydrates :—Gluten and bran bread, bread and biscuits made of almond or cocoa-nut flour. Ordinary bread toasted is very frequently given, and may constitute the carbohydrate element of the diet of our patient; it is often preferable to gluten bread, which is tasteless and uninteresting, and causes the mouth to become tender and painful.

The following wines and spirits may be taken :—Unsweetened Moselles and many varieties of hock, claret, dry sherry, and unsweetened spirits.

Nuts, practically of all kinds, excepting chestnuts; oranges and lemons, and most fresh fruits, but in strict moderation.

Tea, coffee, and cocoa; lemonade and other aerated waters (all unsweetened).

The following articles of diet are prohibited :—

Liver, and soups thickened with flour or starch.

The following vegetables :—Potatoes, turnips, artichokes, and practically all starchy vegetables.

Ordinary bread and all farinaceous food-stuffs, and especially rice, maize, and wheat flour, which contain a large excess of carbohydrates.

All sweet wines, and especially those of the port-wine class, beer, ale, and porter.

Fruit syrups, fresh fruit cooked with sugar; excessive quantities of the sweeter fruits uncooked, and honey.

Chocolate and cocoa sweetened, and any aerated waters and other beverages containing sugar.

On glancing over the tables just given it is apparent that a large range of articles of diet may be partaken of by the patient, but it is prudent to keep in mind that of those

es permitted many, such as fresh fruits, should be eaten in moderation. We can make up for the loss of starchy hydrates by giving fat, which is easily provided in the form of butter or of lard in cookery. It is wise to endeavour to regulate the total amount of fluid which the patient drinks, at least to a certain extent. A strictly dry diet is dangerous, and, like, just as a diet with too little carbohydrates, it may lead to a diabetic coma.

Order a healthy but not excessive amount of exercise for the patient, taking into account the age and capabilities of the individual, and also the type of diabetes present. Forbid all mental strain, and obviate worry and anxiety so far as possible. The patient should have a cold bath every morning, and should be dressed warmly, by preference with wool next the skin, and the utmost care must be taken to keep up a free but not excessive daily action of the bowels. There are certain baths to which patients improve greatly, partly by the diabetic régime, and partly perhaps by drinking the waters. Of these the best are Carlsbad, Neuenahr, and Vichy, while in winter our patients often benefit by a few months in Egypt during the colder months of the year at home.

The Medicinal Treatment.—Unfortunately, so long as the pathology of diabetes is in its present state, we can offer no satisfactory statement as regards drugs. Opium in the form of codein, morphine, or other of its alkaloids, has perhaps given the best results. The dose should be steadily increased, to the equivalent of $\frac{1}{2}$ a grain of opium up to 3 grains daily, and in successful cases marked benefit follows; unfortunately, this is an exceptional experience. There is no possibility of giving internal pancreatic secretion in the same way as it is administered naturally, and no pancreatic extract or preparation has yielded any beneficial results. Alkalies are certainly of value and should be freely administered in the form of aerated alkaline mineral waters, or sodium bicarbonate and citrates can be given in fairly big doses.

For the treatment of diabetic coma alkalies are at present of more benefit than anything else. They may be administered as an intravenous injection, consisting of a 5 per cent solution of sodium bicarbonate in water, and over 400 oz. may be thus injected in 30 hours; certainly 2 or 3 pints may be given at one time. The median basilic vein is

usually selected, and the solution should be warmed to blood-heat. Similar or stronger solutions have been used per rectum, but are not so efficacious, and we should remember that this treatment will be more successful if performed *before* diabetic coma actually develops. Arsenic, bromides (sometimes associated with the opium treatment), benzosol (gr. 15-20 thrice daily), sodium salicylate (gr. 15-20 thrice daily), and many other remedies, have been advocated from time to time, but experience does not bear out the special value of any one of these. Cod-liver oil is often a useful agent where emaciation is marked.

The following special symptoms demand a word in conclusion. Excessive thirst can be obviated by giving a little unsweetened lemon juice or some citric acid in water occasionally. Pruritus and eczema are largely prevented by cleanliness, and a sponge soaked in boracic lotion should be used for cleansing purposes after micturition. Remember that excessive fatigue, some cause of worry, and above all constipation, may bring on diabetic coma, and perhaps the best purgative to give is a saline, such as magnesium sulphate, or one of the purgative mineral waters, of which the best are Carlsbad, Rubinât, and Hunyadi Janos.

VII. DIABETES INSIPIDUS

A FORM of polyuria associated with great thirst, but in which there is no sugar and no albumin in the urine. In many cases there is much difficulty in deciding as to the cause of the condition.

Etiology.—It is a disease of childhood or young adult life, but even then it is very rare. There is sometimes a hereditary history of the disease, but more commonly the history is tubercular.

Blows upon the head, excessive worry and anxiety, mental excitement, starvation and physical neglect, alcoholism, syphilis, many acute affections, including influenza, and perhaps not uncommonly exposure to cold, have all been stated to be exciting factors. There is no question that lesions in or near the floor of the 4th ventricle have occasionally produced this disease.

Pathological Anatomy.—It is a well-known fact that Bernard, by puncturing the floor of the 4th ventricle slightly higher point than the so-called sugar puncture, is able to produce a temporary polyuria. While, therefore, one would expect to find in a certain proportion of cases a lesion at or near this particular point, there are many cases in which a post-mortem examination has yielded no definite pathological results. There is no doubt that exposure to cold induces in many persons a very free though temporary polyuria, and it is possible that a few cases may be due to a serious involvement of the vasomotor nerves, although the mode of production is unknown. Certainly the drinking of huge quantities of fluid, and especially of alcohol, has given rise to cases, and in hysteria a polyuria is extremely common.

Physical Features.—The quantity of urine passed by the patient may be from 3 to 10 times the usual amount. The urine is pale in colour, almost like water; the specific gravity is normally low, often not more than 1002-1005, while the quantity of urine passed may exceed the total amount of fluid drunk by the patient. There is no albumin and no sugar, and although inositol (muscle sugar) is sometimes present, this is probably accidental. In certain cases one finds an excess of phosphates, in others an excess of urates; to the former the treatment of phosphatic diabetes has been sometimes applied. In addition to the polyuria and thirst there are other physical features. The patient is emaciated and appears rapidly to lose flesh. The skin is dry. There is nothing worthy of special mention to be said about digestion, but constipation is a frequent accompaniment. Cardio-vascular changes, such as develop in diabetes mellitus, or of the kidneys, are not present.

There are many different types of the disease which might be described. In certain of these the amount of urine passed may be so great as in others, and the degree of emaciation may vary. Probably in cases in which there is no brain injury, or no history of hysteria or other predisposing disease, the polyuria is most excessive, and the thirst and wasting are most marked.

There is sometimes arrest, the polyuria diminishing or ceasing, and the patient improving in general health. Other cases, and especially those which are tubercular, may die, perhaps from the tubercular condition.

Diagnosis.—The absence of sugar differentiates this disease from diabetes mellitus; the absence of albumin and of cardiovascular changes prevents confusion with chronic Bright's disease or primary cirrhosis of the kidney.

The **Prognosis** depends, as already indicated, on the type of the disease present and on the degree of improvement obtained from treatment. Very severe cases seem to resist all drugs, and a fatal termination usually occurs within a matter of months.

Treatment.—Every probable etiological factor must be thought of and, if possible, treated. With this object anti-syphilitic treatment, treatment for hysteria, the prevention of cold by warm clothing, should all have the attention of the physician. Try the effect of gradually limiting the amount of fluid taken by the patient. Treat with preparations of valerian, ergot, and opium, as all of these sometimes do good, and the first certainly in cases akin to hysteria. Nitro-glycerine is another useful remedy. Sometimes electrical treatment appears to be of distinct service.

VIII. OBESITY

SOME persons have a hereditary tendency to stoutness, and in certain families a single member may become unusually stout even from childhood. Stoutness is often the result of excessive eating, but many stout persons eat little, and want of exercise cannot always be accepted as a satisfactory explanation. Perhaps the most constant and well-recognised fact is the tendency of people who have passed middle life to become stout, and this tendency is often a hereditary one. The excessive ingestion of carbohydrates aids in the production of fat.

The **Clinical Features** include evidence of fatty infiltration of the heart and inability for prolonged effort or exercise. Stout people are bad subjects for anaesthetics, and it is prudent when a general anaesthetic has to be administered, to study the sounds of the heart with special care. Stout persons suffer more severely from any pyrexial disease, because their power of resistance and of recovery are below the average.

Treatment.—The dietary should be studied &

late any excess of carotidemia in the form of a
 should be the first of a number of the disease
 of Banting's disease & the disease in the form of
 partaken of. Banting's disease is a disease which
 may be due to a variety of causes. The disease is
 usually of a chronic nature & is characterized by
 a gradual increase in the amount of carotidemia
 in the blood & a gradual increase in the amount of
 carotidemia in the blood.

INTOXICATIONS

I. FOOD POISONING

(PTOMAINE POISONING AND GRAIN POISONING)

It is convenient to divide food poisoning into two groups of cases. In one group the substance eaten is poisonous in itself, and is only partaken of either with intent to kill or by mistake. This group includes poisonous mushrooms, and the flesh of animals or fishes which have before death partaken of foods poisonous to the human subject. The other group consists of cases in which food, not in itself poisonous, acquires poisonous qualities owing to decomposition.

1. MEAT POISONING

This is generally brought about by the eating of decomposing meat, and especially of sausages, pies, and similar partially prepared meat substances. In most of these cases the meat has become tainted, although, possibly, no unpleasant taste may be recognised.

Tinned meat sometimes decomposes, and where, without the application of heat, the jelly about the meat has liquefied, or there is the slightest tainted odour on opening the tin, the contents should be rejected.

The **Clinical Features** of meat poisoning vary considerably, depending a good deal on what organism is responsible for the decomposition of the meat, but more or less gastrointestinal irritation is present in every case. A period of incubation of varying length may first occur, and then the patient complains of colicky pains, with nausea and later vomiting, whilst rigors are not infrequent. The patient feels chilly, and

rehead is covered with beads of perspiration, diarrhoea sets in and becomes very persistent, and faintness or collapse soon follows in severe cases.

Milk poisoning, or poisoning by the decomposition product of milk, is also the result of a micro-organism, and clinical features are very similar to those given above.

Many shell-fish are poisonous, mainly from the development of a toxin in the liver; a good example is the mussel. Fish occasionally fasten on and utilise as food, substances which may prove deadly to man.

2. ERGOTISM, OR POISONING BY ERGOT

Ergot is a fungus, the *Claviceps purpurea*, which attacks grains, and especially rye, and poisoning generally results from infected grain being partaken of in large quantities as food. The black-looking fungus is easily seen on the heads of the rye-crop. The active principles are thought to be sphacelinic acid and cornutin.

Ergotism may produce (1) a *Gangrenous* and (2) a *Spasmodic* type of poisoning.

In the *Gangrenous* form gangrene attacks the extremities, and especially the toes and fingers, while numbness, tingling, sometimes spasms of muscles, and eventually delirium precede the gangrene. Sphacelinic acid has been considered responsible for this type of ergotism.

The *Spasmodic* type is characterised by remarkable convulsive phenomena, including cramps and spasms involving the muscles of the limbs, and coming on after a period of one or two weeks. Death occasionally occurs with delirium, and convulsions are extremely common. This variety is said to be due to the cornutin constituent of ergot, and the pathological changes in the spinal cord resemble locomotor

3. LATHYRISM

is due to the eating of seeds of the common vetch or

It occurs in countries such as Italy and Spain, in which the seeds are used as food. It causes a transverse myelitis with both sensory and motor symptoms, and the pathological lesion is a toxic sclerosis.

4. PELLAGRA

This is due to a disease affecting the maize crop, common in Italy, Spain, and some other maize-growing countries. There is weakness, spinal pain, sleeplessness, and the appearance of a typical rash called the "pellagra erythema." The skin is shed, and considerable suppuration occurs in connection with it. The other phenomena include dyspepsia with diarrhœa, and the nervous manifestations are peculiarly marked in severe cases. No distinctive pathological change has been described, although the disease not infrequently proves fatal.

Beri-beri is held by many authorities to be dependent on some organism or fungus associated with rice, and therefore it should be mentioned in connection with this subject. It is described elsewhere.

Treatment for the Preceding Conditions.—It is most important to remove as rapidly as possible any of the food which has caused the poisoning. If a considerable quantity has passed on into the intestine, a gentle purge may be advisable, especially if diarrhœa has not commenced. Intestinal antiseptics, such as β -naphthol or salol, are often of advantage, especially where organisms constitute the chief danger to the patient. Stimulants may require to be freely administered, and where toxins cause contraction of the pupil, it has been suggested that atropine, which dilates the pupil, might therefore act as a suitable antagonistic agent. Certainly sedatives should be administered where convulsive seizures or spasms occur.

In the case of meat, sausages, and more especially tinned meat, rigorous examination should be made of stocks exposed for sale.

Where a rye-crop is seriously infected with ergot, it ought not to be used for food; and diseased maize is also dangerous.

II. ALCOHOLIC POISONING

1. ACUTE ALCOHOLISM (DELIRIUM TREMENS)

THIS is apt to occur in an alcoholic individual who has been exceeding his usual limits, or who has had a severe strain or

ness such as pneumonia, for which he has been confined. It has been known to develop in persons in apparently health, but who, after months or years of constant excess, are forced to lie up owing to some operation, and in them, eventually, the confinement to bed is largely responsible for the onset of the delirium.

Pathological Anatomy.—There is no very satisfactory one which can be called a pathological result of delirium. In most alcoholic individuals the membranes of the brain are milky in appearance, although such milkiness has been found present in the most abstemious persons. Swelling has also been described and œdema, but it is probable, judging by recent research, that the œdema may occur in death.

Clinical Features.—The patient is sleepless, restless, highly irritable, and complains of terrifying dreams, and his nervousness greatly increases towards night-time. There is a general tremulousness, especially of the tongue and hands.

If the attack is severe, the patient soon begins to talk incoherently, discussing his business matters in an irrational or confused fashion, and passing from one subject to another without any total absence of continuity. He has hallucinations of sight and smell, and sees rats and creeping things of all kinds; when he reaches this stage the attack is at its height.

In the alimentary system one notices the furred tongue and marked dyspepsia, indicated by the total aversion to food. The patient's pulse is full, fast, and soft, later becoming feeble and irregular. His skin is moist and he sweats profusely, while the temperature is found to be 102°, 103°, or may even reach 104° F., falling from time to time with the profuse perspirations just mentioned. The urine is concentrated, and contains a trace of albumin. There are sometimes attacks of delirium which continue until death, and during these the patient may be guilty of all sorts of impulsive and violent acts, while convulsive seizures, epileptic in nature, are means uncommon.

The attack may gradually pass off, or in fatal cases, as a result perhaps of continued excitement, the patient collapses; death may occur from heart failure, from a low type of pneumonia, or from sheer exhaustion. Each attack is more likely to kill the patient than its predecessor, a fact

which should be used as a cogent argument in dealing with drunkards.

Diagnosis.—As a rule there is little difficulty, because the tremulousness, together with the history of the patient's habits, renders a mistake improbable. In *meningitis* and *general paralysis of the insane* there are phenomena present which are absent here.

Prognosis.—Until the patient is really round the corner, it is impossible to make any definite statement; while the want of will-power, from which most alcoholic persons suffer, renders the probability of cure very uncertain.

Treatment.—It is impossible to say whether complete abstinence is better than a gradual diminution in the quantity of alcohol taken by the patient. As a general rule complete abstinence should be insisted on, and the utmost care taken to allay excitement and to prevent the patient doing himself harm during maniacal seizures. He may be either kept in bed with a sheet fastened over his chest, and if necessary a straight waistcoat put on, or he may be placed in a padded room and allowed to exhaust himself without risk of injury. Whichever plan is adopted, it is of the utmost importance to feed the patient regularly and fully. We should give milk with two or three switched-up eggs and strong soup at frequent intervals. Try by the use of bromide of potash (gr. 20-40), chloral (gr. 20-30), hyoscine (gr. $\frac{1}{60}$ hypodermically), or other sedative, to soothe the excitement and restlessness. Care should be taken of the heart, which is so apt to fail, and digitalis or strophanthus must be administered whenever there is the slightest sign of weakening of the pulse. Aromatic spirit of ammonia, spirit of chloroform, and strychnine are invaluable when stimulants are necessary. The patient should be constantly watched, and it is wise as soon as possible to free him from restraint, because restraint impedes the action of the chest, and alcoholic pneumonia is one of the greatest dangers. Lastly, endeavour to keep the bowels open, and to pay attention to the kidneys if the quantity of urine is insufficient.

(2) CHRONIC ALCOHOLISM

There are many ailments which are the result of the constant excessive use of alcohol. All the alcohols act as



poisons, and amylie alcohol or fusel oil is one of the most potent and dangerous members of the group. Alcoholic liquors contain very different amounts of absolute alcohol. Spirits contain, as a rule, from 50-60 per cent, wine, such as port and sherry, 15-20 per cent; light clarets about 10 per cent, and beers and porter from 4-6 per cent. Individuals vary greatly with regard to their liability to suffer from alcohol, and opinions differ in the medical profession as to what should be called the proper use of alcohol in health and in disease. Reference to the pathological anatomy will show the effect of alcohol, at least in excess, but it is improbable that in great moderation it either shortens a man's life or lessens his mental or physical usefulness. The difficulty is to decide what that limit really amounts to, and it certainly varies in different individuals.

Pathological Anatomy.—There is always congestion of the brain in persons who die after great excess of alcohol, and reference has been made to this under acute alcoholism. In chronic drinkers the nerve-cells show very marked degenerative changes. There is vacuolation of their protoplasm, with definite changes in the chromatic substance, and, as noted under peripheral neuritis of alcoholic origin there may be parenchymatous as well as interstitial changes in the peripheral nerves. In addition the cerebral membranes become thickened, and in arteries, kidneys, and heart there is the pathological condition implied by the term arterio-sclerosis, in which the arteries are thickened, the kidneys show primary cirrhosis, and the left ventricle of the heart becomes hypertrophied. Fatty changes are often found in the liver, while common or alcoholic cirrhosis is not infrequently met with in alcoholic subjects. The stomach may show evidence of chronic gastric catarrh, generally of the hypertrophic type.

There are many diseases especially of the nervous system, which depend to a greater or less extent upon alcoholism; it is only necessary to mention as instances epilepsy, the various forms of alcoholic insanity, and possibly general paralysis of the insane.

Clinical Features.—Chronic gastric catarrh with morning sickness is an almost constant feature in the subjects of chronic alcoholism. There are frequent attacks of jaundice, the face becomes bloated, and generally the body is loaded

with excessive fat. Acne rosacea is sometimes the result of alcoholism, and, when present, adds to the disreputable appearance of the patient's face. The legs are often œdematous, and the urine may contain a constant but small amount of albumin. The chief clinical features, however, are associated with the nervous system, and these include the typical tremor of hands and tongue, the irritable and capricious disposition, and the weakening of all mental powers, and chiefly of the will. The patient becomes excessively untruthful, especially when the matter under discussion is the quantity of liquor consumed or the means of obtaining it, and the constant craving for spirits completely overrules any transient resolution towards reform. The worst type of these patients is to be met with in women, who, as a rule, indulge secretly and sometimes in enormous excess. It is in women that one meets most frequently with peripheral neuritis, which, although it seriously cripples the patient for the time being, has the advantage of offering a chance of cure from the alcoholic habit. The wrist-drop and foot-drop, with the muscular pains on palpation, and the history of cramps, should suggest to the physician the possibility of alcoholic neuritis.

The sequelæ of chronic alcoholism are chiefly diseases of the nervous system, and include probably general paralysis of the insane, epilepsy, and alcoholic insanity, while prolonged excess in the use of alcohol greatly interferes with the vitality generally, and may therefore indirectly induce many diseases, such as pneumonia, erysipelas, and tuberculosis.

Diagnosis.—It is easy to recognise an individual who is hopelessly drunk, but to prevent the possibility of error in diagnosis the iodoform test for alcohol in the urine is a good one. If a small quantity of urine be heated with some iodine and caustic potash, iodoform crystals will be precipitated in the presence of alcohol. Generally the odour from the breath and the smell of alcohol in the stomach contents prevent any mistake, although it is well to remember the possibility of *uræmia*, *diabetic coma*, *cerebral hæmorrhage* or *concussion*, giving rise to symptoms closely corresponding to drunken stupor.

It is often excessively difficult to secure a confession from the subjects of chronic alcoholism, because the want of truthfulness already mentioned, and the natural anxiety of the patient to conceal an alcoholic history, obscure the case, and

much tact is necessary in dealing with women in order to arrive at an accurate opinion. The cross-examination of those dealing with the patient, and an effort to trace the source of supply of the alcohol, may be needed before the truth is elicited.

Prognosis.—There is always a tendency for patients who have been alcoholic to relapse, not once, but many times, and probably the greatest danger is the want of will-power, while it should be remembered that the craving for alcohol may become in reality an insane impulse absolutely beyond any rational control on the patient's part.

Treatment.—No treatment is so successful as shutting up the patient from all possible source of supply. This is best done in asylums, where alcoholic patients are taken in as ordinary inmates. The place of alcohol should be taken by strong coffee, but not by the use of opium or other hypnotics, except under exceptional circumstances. Hyoscine (gr. $\frac{1}{100}$) administered hypodermically is an admirable remedy for diminishing excitement and restlessness, while bromides and alcohol are often of great value. The same care should be taken of the diet as was recommended under acute alcoholism, although, as a rule, the patient is not unwilling to eat fairly well.

The gastric condition may be treated sometimes by emptying out the stomach, sometimes by administering small doses of rhubarb root (gr. 2), sodium bicarbonate (gr. 10), and a stimulant, such as powdered ginger (gr. $\frac{1}{2}$). Strychnine, morphia, arsenic, and cinchona are amongst the most useful remedies which we can employ. The much-vaunted gold-cure consists in the injection hypodermically of about $\frac{1}{10}$ gr. of tincture of gold in water. There is no particular virtue in the treatment, and probably much of the benefit is really derived from the care taken of the patient, and the endeavour to restore more healthy conditions of life. Persons who have been subjects of chronic alcoholism and who have been cured should on no consideration be exposed to the risk of relapse, and it is desirable to prevent the use of alcohol in all forms in the lives of individuals who have once been dipsomaniacs.

III. MORPHINOMANIA

(MORPHINISM ; MORPHIA HABIT)

Acute Morphia Poisoning is readily recognised in most cases. The patient passes through a brief period of excitement into deepening coma, and death may result with great rapidity, the breathing becoming more difficult as the respiratory centres are narcotised. The pupils are contracted until immediately before death, when they may dilate.

Chronic Morphia Poisoning is the condition with which, from a physician's point of view, we have generally to do. The drug, whether opium, morphia, or some other alkaloid of opium, may be taken by the mouth, injected hypodermically, or smoked in an opium-pipe. Many persons acquire the habit after taking some preparation of opium for the relief of pain, whilst others, discovering the soothing effects of the drug, acquire the habit in an insidious manner. Opium, if taken in small enough quantity, temporarily stimulates the brain to greater effort, and only produces the sedative and narcotic effects in larger doses. Unfortunately the effect on the patient is by no means trivial or short-lived. The most intense depression, and moral as well as mental deterioration, soon render the opium-eater a miserable and wretched individual. Not infrequently alcohol is taken for the relief of the depression, and in order to produce the desired effect of the opium larger and larger doses of the drug require to be administered. There is much restlessness, itching of the skin, sleeplessness unless the drug is regularly taken, and general discomfort, while the skin of the face becomes sallow and muddy-looking, and the dress untidy and dirty, appearances which readily indicate the habitué of opium. Should the drug be suddenly stopped, the patient becomes intensely excited, very miserable, and the craving almost beyond endurance.

Treatment.—For an acute case of opium poisoning the stomach must be at once washed out, or an emetic given, and afterwards strong coffee administered to the patient. On no consideration should he be allowed to sleep, but he should be made to walk up and down for several hours after the drug

has been removed from the stomach. The faradic current may be applied to the heart, where cardiac failure is threatening, and cutaneous stimulation is often necessary. Strychnine is one of the best remedial agents in opium poisoning.

In cases of chronic poisoning it is wise to withdraw the drug gradually, and, as a rule, to induce the opium-taker to enter an asylum voluntarily for a period of at least six months. During the period of inevitable discomfort much may be done by careful feeding, by the use of strong coffee and similar remedies to keep up the patient's strength, while other sedatives than opium have sometimes to be resorted to for the relief of the restlessness and the insomnia.

IV. COCAINE POISONING

(COCAINISM)

THIS alkaloid, obtained from the *Erythroxylon coca*, is largely used in both surgical and medical practice, and a habit may be acquired similar to morphinism.

The initial stimulant effect is followed by great depression, for which alcohol is frequently taken by the patient. It is remarkable in how short a time the patient deteriorates mentally and physically, and the treatment for a case of cocaineism should consist in seclusion, the patient being in this way unable to obtain the drug. Sometimes the depression stage is so severe as to demand the use of powerful stimulants, and it is during this period that death is apt to occur where an overdose has been administered.

V. TOBACCO POISONING

PERSONS unaccustomed to smoking, and those who smoke too strong tobacco and in too large quantities, may suffer from this kind of poisoning. It produces cardiac dilatation and often irregularity, a very feeble pulse being associated with intense nausea and a tendency to syncope. The respiratory centre is apparently paralysed before the cardiac centre, and for the treatment of cases of actual tobacco poisoning artificial respiration is generally necessary.

VI. METALLIC POISONING

1. LEAD POISONING

LEAD poisoning is apt to occur amongst workers in certain trades, and especially plumbers, painters, glaziers, and lead-miners, while lead in considerable quantity may find its way into drinking water conveyed by new lead pipes or stored in new lead cisterns, and in connection with this it should be stated that soft water is specially apt to become contaminated when in contact with lead. Less frequently lead may be found in wines, ciders, and other alcoholic beverages, and certain hair dyes owe part, at all events, of their colouring properties to lead. Lead may thus enter the system by the alimentary tract, by the lungs, and also by the skin. Some persons possess a peculiar idiosyncrasy to lead, and poisoning may occur at any age.

Pathological Anatomy.—Lead is believed to have a special influence on the production of arterio-sclerosis; it also causes a form of peripheral neuritis, referred to later, and nerve-cells may show degenerative changes.

Acute lead poisoning is extremely rare, and it is therefore only necessary here to refer to the more usual chronic form.

Clinical Features.—(1) Lead Colic.—Colic is perhaps the most common result of lead poisoning. It in no way differs from ordinary colic, unless it be in its duration and the consequent prolonged agony from which the patient suffers. There may be vomiting, and sometimes diarrhoea ensues.

(2) The blue line on the gums, which does not incommode the patient, is an extremely valuable clinical feature for the purpose of diagnosis. It is situated at the margin of the gum, and is due to a precipitate of sulphide of lead by the sulphuretted hydrogen derived from decomposition changes in connection with the teeth. It need hardly be remarked that the lead which forms the sulphide circulates in the blood. The line may be seen for a long time after the source of poisoning has been stopped.

(3) Severe pains in the back and head are not infrequent in connection with lead poisoning.

(4) Lead paralysis, which is described elsewhere under neuritis, and of which four types may be met with.

(5) Anæmia is often severe, and a lead cachexia is not common. A number of observers have noted that the red corpuscles show a peculiar colour-reaction with certain reagents, which is believed to indicate a basophilic degeneration. In most cases of lead poisoning one or other of these characteristic features predominates, and it is rare to find a case in which all are equally marked.

Diagnosis.—Lead is rapidly eliminated by the kidneys, and therefore may be obtained from the urine. The wrist-annule is extremely typical, and the presence of the blue line on the gums often prevents mistake.

Prognosis.—Recovery is probable in most cases in which the condition is recognised and the introduction of the metal into the system promptly stopped.

Treatment.—For the treatment of a case of lead poisoning potassium iodide is the most important remedy, and it should be administered in doses of 10 to 20 grains thrice daily, while calomel sulphate is given every morning before breakfast in a dose sufficient to produce a free motion. By this means lead is fairly rapidly eliminated from the system. Colic caused by lead demands similar treatment to other forms of abdominal painful complaint, fomentations or poultices affording comfort to the sufferer. The removal by lumbar puncture of a little fluid often relieves the extreme pain in the loins and back, and in lead paralysis, treatment by strychnine hypodermically, by massage, and electricity, yields satisfactory results. The patient is always anæmic, and iron should be administered.

PROPHYLAXIS.—Lead piping and lead cisterns should not be employed where the water is soft, unless it has been chemically protected by producing a precipitate of carbonate of lead which forms a protecting skin. Where white or red lead is manufactured or extensively used, the hands should be thoroughly cleansed prior to eating, and where the lead-dust contaminates the atmosphere, a suitable mask should be worn by workers.

2. ARSENIC POISONING

We have only to consider chronic arsenic poisoning here. It may result from arsenical wall-paper, artificial flowers,

or any coloured material used for dress or in connection with the furniture or hangings of a house in which an arsenical dye is used. Apparently arsenic may pass into the atmosphere in solid particles or in organic compounds, formed partly by the action of certain moulds. Recently quantities of arsenical beer have caused much trouble, due to contamination of the yeast, and perhaps also to arsenic in the coal used for heating. According to some authorities alcoholic poisoning is often really arsenical in nature. The commonest cause of arsenical poisoning is the administration of arsenic to patients suffering from pernicious anæmia or chorea, the dose being carelessly increased until arsenical poisoning supervenes.

The **Clinical Features** include nausea and pain in the stomach, puffiness and congestion of the eyelids, and eventually vomiting and diarrhœa. The gums are apt to bleed, and salivation may occur, and later the phenomena of arsenical neuritis referred to under the nervous system may develop.

The **Treatment** consists in the endeavour to remove the drug, and probably potassium iodide is the best remedy. The arsenical paralysis should be appropriately treated.


3. PHOSPHORUS POISONING

Both acute and chronic forms of phosphorus poisoning may be met with in persons working extensively at trades in which phosphorus is largely used, and especially in the making of matches.

Clinical Features.—The symptoms of **acute** phosphorus poisoning are severe gastric irritation, with pain over the liver, and jaundice. The patient rapidly becomes weak, the pulse feeble, and death generally occurs in about a week, often from cardiac failure.

The symptoms of **chronic** phosphorus poisoning are profound ill-health with anæmia, frequently pain over the liver, and especially the development of phosphorus necrosis, commonly called "phossy-jaw." As a result of this the teeth may fall out, and the patient's health suffers seriously.

The **Treatment** consists in the removal of the phosphorus as rapidly as possible. In acute cases an emetic should be given if phosphorus is present in the stomach, and a weak solution of sulphate of copper will form a black phosphide of



copper which is inert, although the remedy is mainly given to induce emesis.

In the more chronic cases careful dieting should be carried out, and the surgical treatment of phossy-jaw must be thorough in order to be satisfactory.

VII. SNAKE - BITE

THERE are many poisonous snakes in hot countries, and in our own land the ordinary viper or adder (*Pelias berus*) is sometimes very deadly.

The active principles of snake venom may be divided into two bodies :—

(1) A coagulable proteid poison which causes disintegration of red blood corpuscles, and also acts directly on cardiac muscle.

(2) A non-coagulable proteid or albumose, which acts specially on the great nerve centres in the medulla, paralysing the respiratory centre and also the nerve-endings in muscle.

Snake venom differs widely as regards the amounts of these poisons present.

Sir Thomas Fraser and Calmette have obtained an antivenene as the result of inducing immunity by repeated graduated doses of venom, and Calmette finds that the anti-venene obtained by using venom of the cobra de capello is capable of counteracting all venoms, including that of scorpions.

Clinical Features.—There is rapid local swelling, with extensive hæmorrhages owing to the disintegrating action of the coagulable proteid, and this is most marked in the affected limb. Cellulitis, and even gangrene, may ensue in cases of very poisonous bites. There is a tendency to heart failure, dyspnœa, and eventually coma and death.

The **Prognosis** depends on the kind of snake which has inflicted the bite ; the strength of the individual bitten ; the part of the body bitten ; whether the bite is on exposed skin, or on skin covered by clothes ; and the possibility of prompt treatment.

Treatment.—Prevent the poison entering the system by applying a ligature above the part bitten, if it be a limb. Try

to suck out the venom from the bite, taking care that there is no lesion of the buccal mucosa. Administer stimulants, and especially alcohol and ammonia, and apply strong liquor ammonia locally. Give antivenene if obtainable, and do so at once.

SUNSTROKE

(HEATSTROKE ; HEAT APOPLEXY ; SIRIASIS)

THIS is a group of conditions attributed to the heat of the sun, exposure to excessive heat not necessarily solar, and especially exposure to heat when there is an undue amount of moisture in the atmosphere.

Etiology.—The predisposing causes are extremely important. Physical exhaustion, unsuitable clothing, an alcoholic habit, and bodily debility, all render the individual more liable to attack. The sun causes sunstroke in hot countries when either the head, neck, or back is exposed to its rays.

Pathological Anatomy.—Decomposition speedily follows death, there is much post-mortem staining of blood-vessels, and there is usually congestion of the brain and cord. It is very difficult to assert that there are definite central changes which can be called pathogenic. The statement has been made that sunstroke occurs in epidemics, and by other authorities that it is endemic in certain regions. It certainly seems more common near the sea-level and in valleys, whereas at high altitudes it is almost entirely unknown.

Clinical Features.—There are two types of cases—those associated with fever, and those without—and both these types might be more elaborately subdivided.

The *Apyretic Type* is less severe ; it consists in extreme exhaustion with threatening of syncope, a subnormal temperature, tendency to vomiting, dilated pupils, and a small, rapid pulse. Some cases show more dyspnoea than others, and profuse sweating is usually a prominent symptom. A subvariety, frequently associated with fainting turns, has been termed syncopal, but the distinction is unimportant.

In the *Pyretic Type* of sunstroke there is hyperpyrexia, and

to this form the term *Siriasis* is specially applied. It may kill in a few moments, or the patient may survive for some hours or days. There is usually excessive weakness, with flushing of the face, an intensely dry skin, and a temperature which rapidly rises to 106°, 107°, or even 110° F. There is marked dyspnoea, and a quick, bounding pulse, while cramps occur in the muscles of the legs, and convulsive twitchings in the muscles of the face and sometimes of the limbs. There may be a trace of albumin and sometimes blood in the urine. Headache, often extremely severe, mental confusion, and even maniacal symptoms are sometimes associated with this type. These cases may rapidly die, or recovery may occur in those which are less severe, but convalescence is slow, and not infrequently, although the patient does recover, he is found to be unable to reside in a hot climate, and to be mentally unfitted for much, if any, strain,—in fact, a degree of insanity is not uncommonly a sequel to sunstroke in those cases which do recover.

The **Diagnosis** is generally fairly simple; the history of exposure to great heat, whether to the sun's rays or in the stokehold of a steamer, together with any predisposing factor in the patient's history, is usually sufficient, while an ordinary *apoplectic seizure* almost always causes a definite and usually a unilateral paralysis.

Treatment. — In the *Apyretic Type* absolute rest is necessary, and the patient should be kept cool, while vigorous stimulation by alcohol, ether, or ammonia is at once resorted to. Strychnine is often of advantage, and ice may be administered.

For the *Pyretic Type* there is probably no remedy so satisfactory as the application of cold water either to the whole body or to the head and back. Iced cloths when obtainable result in rapid benefit. Strychnine and alcohol, and other stimulants, may need to be administered, but are not so necessary for this type. Sometimes the head requires to be shaved and blisters applied. The patient should be given a small dose of calomel and fed on a light diet.

PROPHYLAXIS. — The prophylactic treatment is simple. Persons resident in hot climates should be enjoined to be temperate, especially as regards alcohol. The head, neck, and back must be protected from the sun's rays, and the sun helmets worn in tropical countries are of great value. On

consideration should individuals who have suffered from stroke be permitted to remain in the tropics if they find exhaustion or mental aberration associated with exposure to excessive heat. Probably there is no remedy so valuable for prevention of sunstroke as water, but, unfortunately, it is difficult for troops on the march in a hot sun to find sufficient water to wet the head and back. The proper ventilation of berths in steamers has done much to obviate heatstroke during.

DISEASES DUE TO INTERNAL PARASITES

I. CESTODES

THERE are three important cestodes which in their adult state are found in man, namely, *Tænia solium*, *Tænia mediocanellata*, and *Bothriocephalus latus*. The head and neck, also called a scolex, the body or strobila, and the segments of that body or proglottides, are common to the group. The terminal segments are ripe and contain testes and uterus, and the latter is full of ova, which are generally evacuated. These ova are often ingested by a suitable host, the shell is digested, and a six-hooked embryo set free, which bores through the intestinal wall, and eventually forms a cyst-like bladder or cysticercus (except in the case of *Bothriocephalus*, in which the larval form remains free). When the cysticercus, or larval form, is eaten by man the adult worm develops in the intestine. Usually the head of the worm is situated high up in the small intestine.

(1) ***Tænia Solium*** is common on the Continent, its cysticercus form being found in the pig. It is 8-15 feet in length. The head, which is about the size of a pin's head, has two rows of hooklets and four sucking discs. The genital pores open laterally, and generally on alternate sides in the proglottides. The uterus has about a dozen lateral offshoots which ramify considerably. The ova are $30\ \mu$ to $35\ \mu$ in diameter.

(2) ***Tænia Mediocanellata*** or ***Saginata*** is the usual British form, as its cysticercus is found in the ox. It is 15-20 feet in length. The head is larger than the last and has four sucking discs, but no hooklets. The genital pores open laterally and have no tendency to alternate, and the uterus possesses often as many as 30 lateral branches, which

do not ramify. The ova are slightly larger than those of *Tania solium*, and more oval.

(3) **Bothriocephalus Latus** is common in Russia, the Baltic, Switzerland, and elsewhere. Its larval form occurs in fresh-water fish, especially pike and trout. It is 20-30 feet in length. The head is long, with a long sucking groove on either side, and the segments are extremely broad, possessing a rosette-shaped uterus in the middle, and the genital pore opens in the centre of the rosette. The ova are about twice as large as those of *Tania solium*.

The **Clinical Features** of the different forms of tapeworm are sometimes not distinctive, excepting that proglottides are found from time to time in the stools. The patient may not suffer at all, but colicky pain, dyspepsia, voracious or capricious appetite, and sometimes constipation, may be present. In some rare cases nervous phenomena appear—fits and even mania being noted. *Bothriocephalus* is credited with producing very profound anaemia. Tapeworms probably live for years in the host, and there may be several individuals present at once.

Diagnosis—The position of the genital pore should render *Bothriocephalus* unmistakable, and the branching of the uterus in the more mature segments is sufficient to distinguish the other two commoner tapeworms from each other.

Treatment.—First starve the patient for some hours, give a purge, and follow with a dose of an anthelmintic, such as $1\frac{1}{2}$ -2 drachms of filix-mas in a carminative infusion, such as cinnamon water. It is generally dispensed as an emulsion. Often smaller doses are administered every two hours, and in any case a purge must follow. Given on an empty stomach the remedy is most efficacious, although it may require repeated doses to bring away the head. The head should be carefully searched for in the stools. Koussou, kamala, and other remedies may be substituted for filix-mas.

VISCERAL CESTODES

1. The **Cysticercus cellulosæ**, or cystic form of the *Tania solium*, is sometimes found in man, when he becomes the intermediate host in place of the pig. It is not easy always to discover how the ova enter the stomach, and it has been suggested that in certain cases where the adult tania is present

in the intestine, the proglottides may be forced upwards into the stomach during prolonged vomiting.

The cysticerci only cause trouble when they are found in certain parts of the body, and especially when they reach the brain or cord. There appears to be little evidence of general discomfort during the boring of the embryos through the stomach wall. In the brain or cord the cysticerci may give rise to the manifestations of tumour, or there may be no symptoms at all. In several recorded cases cysticerci have been found in the eyeball.

The cysticerci of *Tænia mediocanellata* have not been found in man.

II. Hydatid Disease.—The adult tapeworm (*Tænia Echinococcus*) occurs in the dog, but is extremely rare. It is only some 4 mm. in length, consists of 4 segments, of which the head resembles the head of a tiny *Tænia solium*, with two rows of hooklets and four sucking discs. The terminal segment is alone mature, and contains several thousand ova.

If the ova are ingested by man, they develop into little six-hooked embryos which bore through the intestinal wall, and may reach the peritoneum, or a blood-vessel, or lymphatic. When finally located it forms a cyst which rapidly grows in size. The cyst wall is formed of a chitinous ectocyst and a granular endocyst or germinal membrane. The cyst contains a watery fluid 1005-1009 specific gravity, rich in sodium chloride. Soon buds form on the germinal membrane and form daughter cysts; from the germinal membrane of these, grand-daughter cysts may develop, and the daughter and grand-daughter cysts become free. From the germinal membrane of all these cysts brood-capsules develop, each of which forms one or more embryo worms possessed of a head which resembles that of the parent, and which can be everted at will. These are called scolices, and these scolices can become adult worms if ingested by the dog. Sometimes the daughter cysts develop outside, and are called *exogenous*, while a rarer form occurs, in which each cyst after separating from the parent is surrounded by a thick capsule, forming eventually a multilocular cyst.

Cysts may be sterile, and many cysts contain hooklets and shreds of chitinous ectocyst, the result of degenerative changes. These cysts may grow to a huge size and cause much trouble. They may suppurate, they may burst into various structures

sometimes where situated in a kidney, into the pelvis of the ureter, and the daughter cysts cause renal colic when attempting to pass down the ureter).

Hydatid disease is most common in Iceland and parts of Australia, where dogs and men are very closely associated. The cysts occur in different organs in the following frequency: 1862 cases, 953 were found in the liver, 163 were in the intestinal canal, 153 in the lung or pleura, 186 in connection with kidneys, bladder, and genitals, and 127 in the brain and spinal cord.

The **Clinical Features** depend on the organ in which they are situated.

Reference is made under diseases of the liver, kidney, and lung to the presence of hydatid cysts in these organs, with the clinical features in each case, but it must be remembered that hydatid cysts may be found in the brain, in connection with the peritoneum, and in almost any part of the body. The fact that the cysts may continue to grow adds to the risk of pressure by the cyst on neighbouring structures, and of suppuration from infection by pus-producing organisms has been recorded in not a few instances.

Diagnosis.—The character of the hydatid fluid described in the opening sentences should, as a rule, prevent any mistake in diagnosis.

Treatment.—The evacuation of the cyst is the one satisfactory method of treatment, and sometimes aspiration is sufficient to effect a cure, while in other cases, and especially when suppuration follows, it may be necessary to freely open and drain the cyst. The introduction of antiseptic or other fluids into the cyst, or the attempt by electrolysis to kill the parasites within, should not as a rule be attempted.

II. NEMATODE WORMS

The worms taper towards each end. The males, always smaller than the females, have a convoluted testis which opens by a common orifice also to the alimentary canal, while the female possesses two convoluted ovarian tubes which open into the vagina, placed anteriorly. Some of the group are oviparous, some viviparous or ovo-viviparous. The embryos in certain

instances pass through a cycle outside or in a different host—in others the ova are swallowed, and the embryos mature in the alimentary tracts.

Oxyuris Vermicularis or **Thread-Worm**.—This worm is about $\frac{1}{4}$ - $\frac{3}{4}$ inch in length, the female being 2 to 3 times larger than the male, and resembles a small piece of white thread. The ova are oval, and measure about .05 mm. \times .02 mm. It is a widely distributed parasite. The habitat is the large intestine from the cæcum to the anus. Auto-infection is common, the ova being introduced into the patient's mouth. The worms often wriggle out of the anus, causing intense itching, and may even enter the vagina in females. They are found in large numbers in the stools, and are seen to move for some time after exit from the body.

The **Clinical Features** are intense itching of anus, sometimes nose picking, colicky pain, and often reflex nervous phenomena, such as restlessness during sleep with grinding of the teeth, occasionally chorea, and sometimes convulsions. The appetite may be voracious.

The **Treatment** consists in repeated enemata of infusion of quassia, salt and water, lime water, or similar remedy, while internally 2-3 grains of santonin will be found of much service. Attend to the risks of auto-infection; a weak mercurial ointment is used locally with benefit.

Ascaris Lumbricoides or **Round Worm**.—The ascaris is a common parasite in many lands, and especially in warm countries. It resembles a reddish earth-worm—is pointed at both ends, with four longitudinal bands and numerous transverse striations. The female is 7-12 inches, the male 4-8 inches in length. The ova, .07 mm. \times .05 mm., are round or oval in shape. They possess a thick, chitinous shell, and are found in large numbers in the stools. The worms vary in number from 1 to 3 in a child, and 2 to 5 in an adult, but 50 or more have been present in one host.

The habitat is the upper part of the small intestine.

The **Clinical Features** vary; often some colicky pain or discomfort, picking the nose, and scratching the anus, not infrequently slight fever, and voracious or capricious appetite, are noted; while the reflex nervous phenomena may be more

ked than in the case of thread-worms, and twitchings, convulsions, teeth-grinding, and restlessness at night may be more or less in evidence. A poison, which possibly is responsible for the intestinal symptoms, has been stated to be formed by the ascaris. Very rarely an ascaris finds its way up into the stomach, and is vomited, or blocks the ductus choledochus. The **Treatment** consists in santonin, 1 to 5 grain doses, to be followed by a purge and preceded by rather low diet. Santonin should be administered with care, as it sometimes irritates the patient, producing yellow vision amongst other unpleasant phenomena, and a night or two should elapse before repeating the dose.

Anchylostoma Duodenale, Dochmius Duodenalis or **Intestinal Worm**.—The female is $\frac{1}{2}$ - 1 inch, the male about $\frac{1}{3}$ inch in length, and they are of a reddish-brown colour. They are numerous and measure .06 mm. \times .04 mm. The mouth of the parasite is provided with sharp hooks or teeth which the worm fixes on to the mucosa. The male possesses an umbrella-like expansion at the tail, distinguishing him from the female.

The worm is common in many warm countries, such as India, Brazil, and Egypt; it also occurs in Switzerland, and is especially baneful to the navvies when the St. Gotthard tunnel was being pierced. It has recently been met with in Hampshire.

The habitat, as the name implies, is the duodenum and small intestine, and there may be numerous worms present.

The **Clinical Features** include dyspepsia with epigastric tenderness, very profound anæmia with its concomitant symptoms, such as languor, breathlessness, and increasing debility, and later vertigo and even syncope, while cedema of the ankles may develop. Colicky pain, diarrhoea, and melæna are common, and death may result if the case is not treated. Eosinophilia has been noted.

Diagnosis.—The ova in the stools render the diagnosis

The **Treatment** should consist of 15 to 30 grains of thymol in sugar paper, to be taken on an empty stomach, repeated in 7 days, and followed by a purge. The thymol may require to be taken again a few days later. Filix-mas is sometimes

preferred. The anæmia demands energetic treatment. The prophylactic treatment consists in guarding the water-supply from sewage contamination, and in destroying the dejecta of infected persons.

Tricocephalus Dispar or **Whip-Worm**.—This worm measures about 2-3 inches in length, the female being slightly larger. The whip-like anterior part of the worm is about $\frac{3}{4}$ ths the length of the rest of the worm, and the posterior and thicker part is coiled up like a spring in the male parasite. The ova, .05 mm. \times .02 mm., have a distinctive little node at either end, and possess a relatively thick capsule.

The worm is widely distributed, and especially in many warm countries.

The habitat is the cæcum and large intestine.

The **Clinical Features** are often *nil*, but it may produce severe diarrhoea.

The **Diagnosis** is readily made from finding the ova in the faeces.

Treatment is often hardly necessary, but thymol by the mouth and also by the rectum should be tried.

Trichina Spiralis.—The embryos encysted in voluntary muscle, and the migration of the embryos from the intestinal canal to become so encysted, form the disease in man.

The adult worm is found in the small intestine, the female measuring 3-4 mm., the male 1.5 mm. in length. The encysted trichina is .5-1 mm. in length, and is contained in a capsule which, after a time, is infiltrated with lime salts.

When trichinous pork (uncooked) is eaten by man, the capsules are digested, the worms maturing in 3 days. Hundreds or thousands of embryos are produced from each female about the 7th to the 9th days, and these embryos pierce the intestinal wall and find their way possibly by the lymph channels to the muscles, and in about 2 weeks become encysted. The cysts may be seen as minute white specks (when lime salts have been deposited) in the muscle. All the muscles may be affected, but perhaps most constantly the abdominal muscles, the diaphragm, and the intercostal muscles. Raising meat to the boiling-point kills the encysted worms,

therefore it is smoked or imperfectly cooked meat which is dangerous. The pig is the infective agent, and the ratio of infested to healthy animals differs greatly, and is certainly usually over 5 per 1000 in any country. Inspection of meat has diminished cases of the disease, but one infected carcass may lead to a local outbreak.

The **Clinical Features** are fever during the boring of the embryos, with abdominal pain, diarrhoea, and nausea, and sometimes these phenomena begin early and go on for 7-10 days. Typhoid has not infrequently been suspected. In the muscles a very definite myositis results, with pain on pressure and tenderness, local swelling, and almost always marked and sometimes general cutaneous oedema. The muscles become stiff, leucocytosis reaching 30,000 with a remarkable eosinophilia usually constant, and skin eruptions such as boils and urticaria sometimes occur. Dyspnoea depending on the involvement of the respiratory muscles, and difficulty in eating, speaking, and walking, due to the same cause, may ensue, and the patient dies after a prolonged period of increasing ill-health.

The **Diagnosis** may be made by examining a piece of the infected muscle, while the worms are found in the stools up to the tenth day or thereby after ingestion of the infected meat.

The blood should be examined for a leucocytosis and eosinophilia. Isolated cases are rare.

The **Prognosis** depends much on the degree of early intestinal irritation, because diarrhoea favours the premature death of the trichina before maturing. The death-rate varies greatly, being from 2 to 25 per cent.

The **Treatment** consists in free purgation if the case is at an early stage, and filix-mas, santonin, or thymol are recommended as vermifuge remedies. General treatment of the myositis is all that can be suggested later. Prophylaxis consists in the systematic and careful inspection of

Filaria Sanguinis Hominis. — There are four or more species of filaria whose embryos may appear in the blood of man. These embryos are all long, 100 μ to 350 μ in length, 4 μ to 12 μ in diameter. They taper posteriorly and show transverse striations. We do not know so much about the adult filaria.

The *Filaria Bancrofti* is 150 mm. in length, the male being smaller, and resembles a whitish horse-hair; the worms are found in the blood-vessels and also in the lymphatics. They may cause no trouble, but as the result of their death filarial abscesses may develop, and they may block the thoracic duct or lymphatic trunks, and cause extensive varicosities of these channels. This may occur in many positions, and rupture leads to lymphorrhagia. If near the kidney, they cause chyluria; if in the groin, large tumour-like masses, and similarly lymph scrotum, and what is probably elephantiasis of the leg are produced. It is difficult to explain the exact conditions under which elephantiasis and kindred affections arise. It may be from injury of the worm or worms, perhaps causing premature discharge of ova, and the ova may cause the blockage. In any case inflammation occurs with great local thickening of skin, and sometimes huge swellings. Where the embryos can enter the blood, *Filaria Nocturna* is found in great numbers. Where the filaria nocturna is prevalent in tropical and sub-tropical countries, it is often found in the blood of a large percentage of the inhabitants, and is probably transmitted from one to another by mosquitoes. This embryo has a long, translucent membrane which projects beyond the worm at both ends. It appears in the peripheral blood-vessels at night from about 6 P.M. to 8 A.M., increasing in numbers up to midnight. In the daytime the embryos are found in the lungs and large internal blood-vessels. In the mosquito a cycle of development lasting from 2-3 weeks occurs; it develops a 4-lipped mouth and 3-lobed tail, grows much larger, and finds its way eventually to the proboscis, ready to pass into a fresh human host, in which it becomes a filaria Bancrofti again.

The *Filaria Diurna* is the embryo of another species which is only found in the blood during the day. It closely resembles filaria nocturna, and is found in the Niger country.

The *Filaria Perstans* is a smaller embryo, found in certain parts of West Africa, and especially the Congo, and has no special nocturnal or diurnal habits.

There are also other varieties of which our present knowledge is somewhat slender.

The **Treatment** is largely directed to the condition resulting from blockage of lymphatics (see Chyluria).

Dracontiasis, Dracunculus Medinensis or Guinea-Worm.

—This worm is common in West Africa, India, Persia, Arabia, Brazil, and elsewhere. It inhabits the connective tissue of man.

The guinea-worm is 1-4 feet in length, and about $\frac{1}{16}$ inch in diameter. The male has not been found. Usually one worm is alone present. It enters by the stomach and finds its way into the subcutaneous tissue, and thence head first to the region of the ankle. The head pierces the skin, and a blister forms, which bursts, leaving an ulcer, in the floor of which the head of the worm may be seen. Meanwhile the uterus has become filled with hundreds of developing embryos, and it discharges them, when ready, in a whitish fluid. These embryos are .6 mm. in length, and they develop in the fresh-water cyclops. Once the uterus is empty, the worm can be withdrawn by winding it day by day on to a piece of wood. The worm cannot be removed at an earlier period unless the body is injected with 1 in 1000 perchloride of mercury, and its fracture with, in consequence, a portion of the worm left *in situ* is apt to lead to serious inflammation. It is prudent, therefore, to await the rupture of the uterus, or to carefully inject with perchloride of mercury before attempting the withdrawal of the parasite.

III. DISTOMIASIS

WHILE there are a number of different forms of the distomum, there are only three which demand reference here.

1. The **Distomum Hepaticum**.—Of this group there are several subvarieties met with in different countries, and the parasites vary considerably in size, being from 10 to 20 mm. in length, and 2 to 5 mm. in breadth. The ova are proportionately extremely large, often measuring $30\ \mu \times 15\ \mu$. The phenomena produced by the presence of the distomum vary considerably, but pain over the liver, diarrhoea, and occasionally jaundice have been described. The ova may be readily recognised in the stools.

2. The **Distomum Westermanii**.—This parasite attacks the lung and bronchial tubes, and causes a disease more common in the far East. The parasite induces attacks of

hemoptysis varying in severity, with generally a considerable degree of bronchitis.

3. **Bilharzia Hæmatobia.**—The worm is one of the few members of the group which have the two sexes separate. The female is carried in a special canal inside the male worm. This parasite is specially common in Africa, in parts of India, and in Egypt, and affects the kidneys, ureter, bladder, and rectum. The adult worms are found in the veins of the kidney, although they may also be present in the portal circulation. We do not know how the parasite enters the body, but it has been thought that the embryos may be drunk with water or eaten adhering to water-cresses. The worms are first found in the portal veins but eventually reach the veins in the neighbourhood of the kidney and bladder. The ova, about $\frac{1}{80}$ inch in length, have a peculiar sharp spine, which enables them to pierce their way through the tissues and to reach the bladder, pelvis, or ureter; to a less extent they also pass into the bowel and escape with the stools.

Clinical Features.—The presence of the adult worms produces no trouble, but the ova piercing through the urinary mucous membrane cause considerable hæmorrhage and much irritation, and the ova in the bladder may become the nuclei of stone in the bladder. In the rectum they cause hæmorrhage and often considerable tenesmus.

The **Diagnosis** consists in the recognition of the typical spined ova in the urine and feces.

The **Treatment** consists in the administration of turpentine in fairly full doses, and sometimes the use of filix-mas but neither remedy is very successful. It is impossible to do anything to destroy the adult worms in the veins.

DISEASES OF THE ALIMENTARY SYSTEM

I. DISEASES OF THE MOUTH AND TONGUE

(1) **Stomatitis**

THERE are many organisms found in the mouth, some of which enter with the food; others are associated with dental caries, and others with slight and trivial inflammation of the tonsils. The fur of the tongue consists largely of food and desquamated epithelial cells; but there are many organisms present in addition, and it is known that even the pneumococcus and diphtheria bacillus may exist in the mouths of perfectly healthy persons.

(a) **Simple Stomatitis.**—A slight inflammation of portions of the mucous membrane, causing redness, heat, and dryness of the mouth and furring of the tongue. It may be due to the action of bacteria or to irritants of various kinds, and it is associated with certain of the eruptive fevers. In children it may cause a trivial constitutional disturbance. The affection is often associated with dyspepsia, the use of too hot tobacco, the ingestion of over-spiced food, and in children it commonly accompanies dentition. There may be a considerable amount of discomfort rather than marked pain.

The affection is rapidly cured when the cause is removed.

Treatment.—Remove the cause, give the patient cold or tepid articles of diet, and paint on the inflamed surface either 1/3 or 4 grain to the ounce solution of silver nitrate, or the pharmacopœial solution of borax in glycerine.

(b) **Follicular Stomatitis, Aphthous, or Vesicular Stomatitis.**—Small raised vesicles, with a reddened area around, appear in varying numbers on the lips, tongue (tip

and edges), and gums ; they rupture, and leave small and very painful ulcers.

Etiology.—Usually indigestion is directly responsible for the follicular ulcers. They are common in gouty persons, and also in children under the age of three.

The **Clinical Features** are pain, especially on eating or drinking anything hot, and often marked salivation. The ulcers heal rapidly with treatment, and only in rare cases are they troublesome.

Treatment.—Careful attention should be paid to any evidence of indigestion, and very generally an alkaline purgative gives relief. In children a dirty feeding-bottle, or some similar cause, should be sought for. Treat the condition locally by painting on silver nitrate solution similar in strength to that mentioned above, or by touching the ulcers with a modified lunar caustic pencil. In any powerful application to the ulcers care should be taken to dry the mouth beforehand. Various antiseptics, and especially chlorate of potash, borax and glycerine, or permanganate of potash, are often beneficial when used as a mouth wash.

(c) **Ulcerative Stomatitis or Foetid Stomatitis.**—A more extensive ulceration than the last, beginning at the margin of the gums, with greyish-white ulcers, often causing loosening of the teeth and even necrosis of the alveolar margin of the jaw.

Etiology.—The disease occurs in badly nourished children, after the first dentition, and especially where there is a scorbutic element present. It may be due to dental decay, but the fact that it has been found in epidemic form suggests a specific organism, although no such organism has as yet been isolated.

Clinical Features.—There is pain on chewing, and the swollen gums bleed readily. There is copious salivation, and the breath is foul-smelling. Generally some pyrexia is associated. The condition may go on for a long time, even for months, and produce considerable cachexia. There is painful enlargement of the neighbouring lymphatic glands.

Prognosis.—The disease is rarely fatal, but some of the teeth and part of the jaw may be lost.

Treatment.—The fact that a specific organism is almost certainly present suggests the necessity of using strong anti-

ics. The mouth may be washed out with saturated rate of potash solution, a solution of permanganate of ash, listerine or similar mouth wash, while the patient should have good diet and plenty of open air, and probably it would be well to add the use of lime-juice in most cases.

(d) **Parasitic Stomatitis, Thrush, or Muguet.**—A condition due to the presence of a fungus called *Oïdium albicans*, though in many cases other fungi or bacterial agents are present. Whitish patches form on the tongue, and, on rubbing, a raw tender surface is exposed which often bleeds. It is most common in infants, is generally the result of a dirty nipple or other want of cleanliness, and causes local heat, pain, loss of appetite, and very frequently diarrhoea. Occasionally the patches are situated on the oesophagus or on the stomach, but this is rare. In adults we associate the appearance of thrush with patients in the later stages of any long disease, and especially phthisis, and it may occur in a prolonged fever. In such cases it causes much discomfort and greatly interferes with the digestive processes.

Treatment.—The condition is very amenable to treatment. It is well to give an alkali, such as bicarbonate of potash with water, and to treat the mouth locally with the pharmacopœial solution of zinc and glycerine, silver nitrate solution (10 grains to the ounce), or, in adults, the glycerinum acidi carbolicum diluted in equal parts of glycerine.

(e) **Cancrum Oris, Noma, or Gangrenous Stomatitis.**—A rapidly spreading gangrene, due to the presence of one or more specific organisms, and commencing generally near the angle of the cheek and gum. It is practically confined to children between the ages of 2 and 5, and very rarely follows an attack of measles. In a few cases adults have occurred. It runs a rapid course, generally involving the jaw and causing intense fœtor, salivation, and a great amount of pain, while vomiting and diarrhoea frequently indicate absorption of bacterial poisons. The disease is almost fatal in most cases.

The **Treatment** consists in the application of strong antiseptics such as fuming nitric acid, to the site of the gangrenous area, especially when, as generally happens, it has opened on the cheek. Give iron in large doses and chlorate of potassium. Feed well, and order wine and other stimulants.

Possibly patients in whom the disease proved fatal might have been saved if the treatment employed had been more radical at the outset.

(*f*) **Mercurial Stomatitis.**—Certain patients are specially susceptible to the smallest dose of mercury, and a peculiar idiosyncrasy to the drug is apt to develop in persons suffering from Bright's disease. Patients who are getting large doses of mercury, cinnabar miners, and those who work at trades in which mercury is much used (mirror-makers and thermometer-makers), frequently develop mercurial stomatitis.

Clinical Features.—The gums and tongue are red, raw, and swollen, the teeth loosen, and there is always profuse salivation with dreadful fetor, and sometimes necrosis of the jaw follows.

The condition is amenable to treatment, although it may cause much temporary suffering.

Treatment.—Stop the mercury at once and give iodide of potassium with a saline purge. Atropine has been recommended, but it is of greater importance to keep the mouth aseptic, and to give the patient good nourishing food, which, however, should be partaken of cold or lukewarm.

(2) **Glossitis, Inflammation of the Tongue**

The tongue may be inflamed as the result of an irritant, and perhaps most commonly from an injury, a septic wound, or the sting of a wasp or bee.

The swelling may be so excessive as to prevent swallowing, and the glands below the jaw are secondarily involved. This condition may be very serious or very trivial; in most cases the treatment consists in endeavouring to allay the inflammation, while in certain instances incisions may be necessary so as to reduce the inflammatory oedema.

(*a*) **Ichthyosis Linguae, or Leucoplakia Buccalis.**—This consists in whitish pearly patches, which appear on the dorsum of the tongue and resemble corns in appearance. The disease is due to tobacco, possibly to syphilis, and it yields in most cases to suitable treatment.

Treatment.—Order the patient to take the food cold, and to stop all over-spiced or irritating articles of diet, and paint the patches with silver nitrate solution from time to time.

(b) **Tuberculosis of the Tongue.**—A rare condition, almost invariably associated with a late stage of pulmonary phthisis. The ulcer which eventually forms has whitish thickened edges and is difficult to distinguish from a gummatous ulcer, except by the fact that it does not yield to mercurial treatment.

Little can be done by way of radical cure, but the ulcer should be scraped and the mouth washed out with an antiseptic solution.

(c) **Syphilitic Ulceration of the Tongue.**—Many authorities consider that *ichthyosis linguae* is of syphilitic origin. Typical ulcers, however, affect the sides and the dorsum of the tongue near the tip, and they have somewhat thickened edges and a whitish floor. They may produce marked scarring, and are often associated with gummata. A trivial syphilitic ulceration may be associated with early secondary syphilis.

Treatment.—Give antisyphilitic remedies, including both mercury and iodide of potash, while a mercurial lotion of 1 in 5000 corrosive sublimate should be used as a mouth wash.

(d) **Cancerous Ulceration of the Tongue.** Almost invariably cancer of the tongue is a squamous epithelioma originating at the root of the tongue and causing gradual infiltration, and usually before a definite ulcer has formed, it has involved the neighbouring lymphatic glands.

The **Treatment** consists in the removal of the tumour.

The tongue may also be involved by warts and fibrous tumours which usually cause little pain, but which should be treated surgically, as such growths may eventually become malignant in character.

(3) The Tongue in Dyspepsia

In many cases of dyspepsia the flabby, swollen tongue, thickly coated with fur and often showing marked furrowings on the dorsum and edges, is an important sign of prolonged and persistent dyspepsia. The tongue usually cleans first at the tip and edges, and the appearance of the tongue, especially in relationship to its covering, often indicates some gastric disturbance. In acute gastric catarrh the tongue is thickly furred, the fur being white in colour and moist, but the tip and edges are bright red.

(4) **The Tongue in Fever**

When there is any diminution in the secretion of saliva, such as usually results from pyrexia, the tongue becomes dry, and if it is furred, the fur is often of a brownish or yellowish colour.

In scarlet fever, where there is inflammation of the tongue as well as inflammation of the skin generally, that organ is swollen and is covered with a white fur, through which the swollen fungiform papillæ show distinctly as bright red spots. At a later stage of the fever the fur is shed, and the tongue becomes bright red in colour all over, and often dry and glazed. In all febrile conditions, in which the patient lapses into what is described as the typhoid state, the tongue may either be covered with brownish fur, or may be red and shining.

Tremulousness of the tongue is a symptom of many diseases; it is common in the typhoid stage of fever, in delirium tremens, and in other serious conditions.

Paralysis of the tongue will be referred to under Diseases of the Cranial Nerves.

II. DISEASES OF THE SALIVARY GLANDS

(1) **Excessive Salivation or Ptyalism**

THIS condition has been mentioned as a feature of many forms of stomatitis. An excessive flow of saliva follows the use of certain drugs, in particular mercury, tobacco, and pilocarpine. Ptyalism is not uncommon as a feature in certain mental cases, in small-pox, and occasionally in pregnancy. The only treatment consists in an endeavour to remove the cause of the condition where possible.

(2) **Xerostomia**

Wherever there is an excessive drain either from the kidneys, bowels, or skin, there may be a diminution in the flow of saliva, but the disease called xerostomia is very rare; in it there is no saliva, the mucosa is red and shining, and bread chewed by the patient becomes a dry powder. No treatment appears to avail in such cases.

(3) Inflammations of the Salivary Glands

Mumps or parotitis is an infective inflammation of these glands, and is described under infectious diseases (p. 46), but must be remembered that the salivary glands may swell the result of neighbouring inflammations, and in certain continued fevers suppuration occurs in these glands without, apparently, any very satisfactory reason.

In injuries to the peritoneum, and in abdominal and pelvic lesions of different kinds, the parotid may become inflamed.

(4) Salivary Calculi

These may form in the ducts of the glands, and usually consist of carbonate or phosphate of lime. A fistula or abscess may result.

(5) Cysts and Tumours of the Glands

A ranula is a cystic swelling due to blockage of one of the gland ducts. Gaseous tumours have been caused in the ducts and glands as the result of certain trades, such as bellows-blowing, playing on wind instruments, etc., but they are rare.

Tumours of various kinds are sometimes present, chiefly of the parotid gland, and they may be fibrous, malignant, or cystic. These affections belong more properly to the domain of surgery.

AFFECTIONS OF THE PHARYNX AND TONSILS

It is remarkable how often the pharynx is inflamed while the tonsils, comparatively speaking, escape, and *vice versa*. The tonsils are situated, however, in the pharynx, and may be inflamed with the rest of the pharyngeal mucosa. It should be remembered that the tonsils are lymphoid tissue, and as their cells act as phagocytes and should destroy organisms.

They may in certain cases either fail in this function and become themselves attacked, or else the phagocytic cells may be overcome by organisms which are too strong for them, and in this case infective organisms either attack the surface of the tonsils, penetrate and set up inflammation in their substance.

(1) **Acute Pharyngitis, Simple Inflammatory Sore Throat**

Catarrhal inflammation of the whole pharyngeal mucous membrane, often including the tonsils.

Etiology.—Cold and exposure are frequently the exciting causes, and especially in rheumatic or gouty patients.

Clinical Features.—The typical features are: heat, dryness in the throat, soreness on swallowing, and hoarseness if the larynx is involved. It often begins with coryza, and may be accompanied by slight fever, while aching in the back and limbs is very generally present. There may be a short and constant cough with the hawking up of a little mucus. The Eustachian tubes, if interfered with, give rise to deafness, and the neighbouring lymphatic glands are often swollen. A varying amount of malaise, largely dependent on the degree of pyrexia present, adds to the general discomfort of the patient.

On examining the pharynx it is seen to be reddened, the uvula often showing this very markedly, and there may be a little glairy mucus present in the later stages.

In a few days the condition subsides.

The **Treatment** consists in giving salicylate of soda (if a rheumatic case), quinine, guaiac resin, or Dover's powder. Quinine is admirable in fairly full doses (gr. 3-10), and a single dose of Dover's powder, following a hot bath or foot-bath, frequently aborts a severe pharyngitis. Tincture of aconite is another favourite remedy in cases with much feverishness, and should be given in 1 or 2 minim doses every 2 hours. Inhalations of steam afford the greatest relief, either alone or impregnated with pine oil, creosote, guaiacol, or other volatile antiseptic.

(2) **Septic Pharyngitis or Hospital Throat**

An acute form of pharyngitis in which the tonsils almost always participate, and characterised by patches of membrane due to suppuration of the lymph follicles.

Etiology.—Decaying organic matter and defective drains are often responsible, but individuals exhausted by hard work are apt to be affected by organisms obtained from many sources, and organisms which in persons above par would probably do no damage.

Clinical Features.—The throat is intensely inflamed and very painful, and patches of whitish-grey membrane, which is easily removed by scraping, are seen on the tonsils and larynx. The neighbouring glands are swollen, and there is always a high temperature and marked malaise with pains in the neck and legs. In suspicious cases examine a swab for the diphtheria bacillus. The patient is often very sharply ill, but with suitable treatment improvement is rapid.

Treatment.—Apply at once to the patches either the 1 per cent. solution of mercuric iodine diluted with 2 parts of glycerine, or a 1 in 3000 solution of corrosive sublimate. Gargle the throat with permanganate of potash solution, a saturated solution of potassium chlorate of potash, or boracic acid, or other suitable antiseptic. Give food and drink well, order stimulants freely, and in severe cases give morphine to suck.

(3) Chronic Pharyngitis

A more chronic inflammation affecting the naso-pharynx and the posterior pharyngeal wall.

Etiology.—It is the result of cold and damp, of excessive exertion or strain in hawkers, clergymen, etc., and arises from the immoderate use of certain irritants, such as tobacco and alcohol.

Pathological Anatomy.—There are often dilated venules and a granular-looking posterior pharyngeal wall.

Clinical Features.—No constitutional phenomena are present. There is a tickling cough with tenacious and limited sputum, and, as a rule, a relaxed uvula, which tends to hang down on the tongue. Several varieties of the condition occur and have been given separate titles, such as **Pharyngitis sicca** with total want of secretion; **Follicular pharyngitis**, in which the follicles specially suffer; and **Ulcerative pharyngitis**, which needs no definition.

The **Prognosis** depends on the possibility of resting the throat and the success of treatment.

Treatment.—Stop any causal condition present. Paint the throat with nitrate of silver solution (gr. 3-10 to the oz. of water) or chloride of zinc (gr. 5-10 to the oz. of water). Potassium iodide or chlorate of potash in tabloid form is also of use. Where there are enlarged follicles try to destroy them with the galvano-cautery or with strong silver nitrate solution. A warm, dry climate is advisable, and many spas

are recommended as much for the rest and change as for any special benefit likely to be obtained from a particular mineral water, unless perhaps in rheumatic or gouty subjects. Painting with Mandl's solution, the various strengths of which are given below, is often successful in cases in which all else has failed.¹

(4) Phlegmonous Pharyngitis

A diffuse suppurative inflammation involving the deeper tissues, and generally spreading from some superficial focus. It may be due to the streptococcus of erysipelas, and it is a grave and fortunately rare sequel of scarlatinal pharyngitis. It is a typically septic condition, and it rapidly proves fatal in most cases, and perhaps especially where it spreads and involves the larynx.

The **Treatment** must consist in an attempt to combat the tendency to septicæmia; and free incision, where possible, should be promptly carried out.

(5) Tubercular and Syphilitic Ulcerations of the Pharynx

Tubercular ulcers are rare, and, as in tubercular ulceration in the mouth, are generally associated with grave pulmonary phthisis. They are often very painful and may render swallowing difficult. **Syphilitic** ulcers may be secondary, and if so, are shallow, or they may be tertiary and deep-seated. Syphilitic ulcers are rarely painful.

(6) Acute Tonsillitis or Quinsy

An inflammation of one or both tonsils with occasional suppuration.

Etiology.—It is due to cold and wet, but it is probable that in many cases organisms emanating from decaying matter, perhaps, especially from drains, may enter the crypts of the

¹ Mandl's solutions. Three strengths—

| R. | | (1) | (2) | (3) |
|----------------------|-----|-----|-----|-----|
| Potassii iodidi | gr. | 25 | 50 | 75 |
| Iodi puri | gr. | 6 | 12 | 20 |
| Glycerini | ℥ | 1 | 1 | 1 |
| Olei menthæ piperitæ | ℥ | 5 | 5 | 5 |

sils and there set up acute inflammation. It bears a very close relationship to rheumatic fever, and the tonsils are apt to be involved in scarlet fever, diphtheria, and other infective diseases. There is a distinct predisposition to the affection in certain families and individuals, and persistent enlargement of the tonsils, often associated with the presence of adenoids, appears to predispose to acute attacks of tonsillitis.

Pathological Anatomy.—The tonsillar crypts become filled with a caseous material containing micro-organisms. When an abscess forms it is often peritonsillar in position, and generally points towards the mouth.

Clinical Features.—The onset of tonsillitis is sudden and is generally associated with a rigor, with aching in the back and limbs, and local pain on swallowing, which shoots up to the throat.

The breath is bad, the tongue foul and furred, and on examination the tonsils, one or both, are seen to be red, inflamed, and swollen. They may touch in the middle line, the uvula being in contact with both tonsils. The neighbouring lymphatic glands are often swollen; there is frequently delirium, and almost always a degree of malaise. The temperature varies from 102° to 103° F., and is generally more raised in cases which go on to suppuration and in septic tonsillitis, in which the tonsils specially suffer. There is infrequently superficial ulceration, especially where the tonsils come in contact, and the voice has a distinctive hoarse or nasal tone, while in many cases the tonsillar inflammation interferes with the orifice of the Eustachian tube, causing deafness on the affected side. These phenomena are much more marked when suppuration has occurred. Recognition of an abscess may be made by digital examination—the finger to be introduced into the mouth being passed behind the tongue and in contact with the lower teeth. Very rarely does an abscess open into an artery, and only in exceptional cases does the pus infiltrate the tissues of the neck.

Diagnosis.—*Diphtheria*, with which the disease may be confused, has a distinct membrane which may be absent in tonsillitis, and in the membrane the presence of the diphtheria bacilli should be easily demonstrated, while albuminuria is common in cases of diphtheria.

Prognosis.—Generally rapid recovery results, but with a tendency in certain persons to recurrence during cold, damp

weather. Only in very rare cases does a tonsillar abscess give rise to serious trouble, and then it is usually due to neglecting early incision.

Treatment.—Give ice or iced barley water, and paint the inflamed tonsils with a 10-20 per cent solution of cocaine, or with carbolic acid and glycerine; steaming and poulticing the throat is useful where an abscess is threatening. Invariably give a purge, and by preference salts or compound jalap powder (gr. 20-30); at an early stage quinine in 3-4 grain doses, guaiac resin (3 grains in lozenge form), and tincture of aconite (m 2-3) are often of great value.

When suppuration has occurred it is better not to delay incising the tonsil. After painting with cocaine, or giving the patient ice to suck, take a Syme's abscess knife, the edge of which should be guarded with sticking plaster up to within about $\frac{1}{2}$ an inch from the point, and make a vertical incision, remembering the position of the internal carotid artery. Hæmorrhage after the operation is rare, and ice will usually check it if excessive. After most cases of tonsillitis the tonsils should be painted with Mandl's solution.

In rheumatic patients give salicylate of soda or other agent of the same group.

(7) Chronic Tonsillitis

Chronic enlargement of the tonsils, not infrequently associated with hypertrophy of the adenoid tissue in the nasopharynx.

Etiology.—It is very common in children, especially if they are unhealthy; it sometimes follows an acute attack, or it may be chronic from the outset. It may be congenital; and it frequently is a sequel to diphtheria.

Pathological Anatomy.—There is thickening of the fibrous stroma of the tonsil, and often increase of lymphoid tissue. The tonsils remain permanently swollen, are palpable externally, and may interfere very seriously with the orifices of the Eustachian tubes, thus causing deafness.

As a result of the condition in childhood the chest is badly developed and narrow, and generally the patient's mental growth appears to be seriously impeded, if not checked. In not a few cases of this disease it is found that the enlarged

tonsils are either tubercular or are associated with tubercular glands, and there is a distinct tendency to the development of bronchial catarrh.

Clinical Features. - The visible and palpable enlargement of the tonsils, associated often, as already stated, with adenoids in the naso-pharynx, causes loud mouth-breathing with snoring at night, a peculiar change in the voice, deafness, and a special liability to subacute attacks of tonsillitis and to colds in the head. The child constantly keeps its mouth open, and may have more or less cough, while nocturnal paroxysms of dyspnoea sometimes occur. The existence of mental backwardness has already been noted. When a patient reaches the age of thirty, the tonsils often cease to give trouble.

Prognosis. The tonsils should be removed, even although there is the probability of their subsidence in adult life, and, in most cases, if this is thoroughly done, physical and mental development, together with freedom from attacks of tonsillitis and coryza, may be assured.

Treatment.— Remove the tonsils either with an ordinary bistoury or with the guillotine, and apply astringents or ice to arrest the hemorrhage if excessive. Give in all cases in children cod-liver oil, iron, and other tonics; it is also wise after operation to use an antiseptic gargle for some days. Adenoids in the naso-pharynx should also be operated on if they interfere with breathing.

IV. AFFECTIONS ASSOCIATED WITH THE ERUPTION OF THE TEETH

TEETHING is likely to cause trouble during the *first* dentition, and especially in delicate children who are rickety or tubercular.

The chief **Clinical Features** are swelling and tenderness of the gums, stomatitis, gastric catarrh and diarrhoea, and very frequently eclamptic seizures or convulsions. Prolonged ill-health may result, the child becoming fractious and sickly. Improvement commences once teething is over.

With the *second* dentition there is usually no trouble, but should the patient be delicate, or the eruption of the teeth

be accompanied by much local pain and swelling, nervous phenomena may develop, and especially reflex cough, irritability of temper and asthma, while sometimes gastro-intestinal disturbances are present.

Treatment.—In the infant it is usually only necessary to pay attention to the hygiene of the mouth and to the condition of the stomach and bowels, but if convulsions have occurred, the gums should be lanced.

For trouble during the second dentition similar treatment may be adopted.

V. DISEASES OF THE ŒSOPHAGUS

(1) **Acute Œsophagitis**


AN acute catarrhal inflammation of the œsophagus.

Etiology.—It may be the result of (1) injury; (2) the extension of a catarrhal pharyngitis, and especially in certain continued fevers, such as diphtheria; (3) irritation due to the presence of a new growth or a foreign body; and (4) the swallowing of a corrosive substance. Any focus of irritation in the neighbourhood of the œsophagus may set up œsophageal catarrh.

Clinical Features.—There is pain on swallowing, which may become so excessive as to cause complete inability to take food, and there may be spasm with regurgitation of what has been swallowed. The pain is marked along the line of the œsophagus, and is felt in the neck, under the sternum, and to a less extent in the back. In diphtheritic cases the presence of a membrane adds to the obstruction.

Diagnosis and Prognosis.—The diagnosis depends largely on the recognition of the nature and cause of the inflammation present, and the prognosis on the possibility of successful treatment.

Treatment.—Sucking ice and the administration of soothing drinks are most important, while rest to the inflamed surface may be obtained by feeding by the bowel, and limiting as far as possible the swallowing of saliva by the use of sedatives.



(2) **Stricture of the Œsophagus**

A. Spasmodic Stricture.—Spasmodic contraction of the muscular fibres of the œsophagus.

Etiology.—It is common in hysteria, and is associated with the neurotic habit, and sometimes with gout. It is one of the symptoms of hydrophobia.

Clinical Features.—There is inability to swallow solid or sometimes fluid food. There may be pain and generally regurgitation of food. Great relief is experienced by the passage of a bougie, which, although stopped by the spasm, is eventually passed with ease, and produces a remarkable effect, perhaps partly mental, in alleviating the condition.

The **Diagnosis** is directed towards the discovery of any possible organic cause for the condition, and it must be remembered that with an organic stricture there is a great element of spasm superadded. Malignant stricture and cancerism should both be thought of.

Prognosis.—Recovery generally follows treatment.

Treatment.—The occasional passage of a bougie with general tonic treatment is usually sufficient. Any constitutional element in the case, such as gout, should be treated.

B. Simple Stricture.—A chronic thickening of connective tissue, causing narrowing of the œsophagus.

Etiology.—A rare condition, unless as the result of following an irritant or corrosive fluid, when it follows from the process of healing or cicatrization. Alcohol is said very occasionally to produce this condition. It should be remembered that aneurisms, enlargement of the thyroid, pericardial effusions, glandular swellings, and tumours of all kinds, may exert pressure on the œsophagus.

Clinical Features.—There is increased difficulty in swallowing with an element of spasm, the degree of which depends on the amount of obstruction. There may be retention of food, and very frequently semi-fluid or fluid passes down the gullet more easily than solids.

The **Diagnosis** and **Prognosis** depend on the recognition of the cause of the constriction and the possibility of doing anything to relieve it.

The **Treatment**, in cases where there is actual stenosis due to cicatrization, consists in an attempt at dilating the œso-

phageal stricture, while the element of spasm may be combated by such sedatives as dilute hydrocyanic acid (m 3). Very occasionally surgical interference may relieve pressure from tumours, but it depends entirely on the nature and site of the tumour.

C. Malignant Stricture.—The commonest form of tumour is the squamous epithelioma, although much difference of opinion exists as to where the tumours are generally situated, and each third of the œsophagus has been favoured by different authorities as the commonest position for such a tumour. Probably the upper and the lower thirds are more frequently affected than the middle third, and malignant stricture is very common just at the bifurcation of the trachea. The growth may spread in an annular fashion round the œsophagus; it may infiltrate the walls, and extend upwards or downwards for a distance of 2 or 3 inches. The results of such a tumour may include the actual narrowing of the lumen of the gullet, and perforation through the wall of the œsophagus, often into the left bronchus, while infiltration of the muscle of the œsophageal wall interferes with normal peristaltic movement. Added to these, the presence of a tumour gives rise to spasm when food reaches the stricture, and dilatation of the œsophagus may occur above the stricture, due to food tending to accumulate; such a dilatation may empty itself reflexly from time to time. Perforation into the bronchi or trachea must inevitably cause septic broncho-pneumonia, while perforation into the pleura or elsewhere usually leads to serious and often fatal sequelæ. Secondary deposits are common, involving the glands and other neighbouring structures, and thus death may be brought about, not merely by the starvation of the patient from blocking the œsophagus, but in many other ways.

The chief **Clinical Features** have been already indicated—emaciation and dysphagia, with frequently regurgitation of food, while often the attempt at swallowing produces excessive pain. Generally the food regurgitated is found to be alkaline, indicating that it has not entered the stomach.¹ The patients in whom one finds the disease are usually past middle life.

Evidences of the septic broncho-pneumonia, empyema, and other results of perforation may follow as the disease advances.

¹ This statement is not always accurate, because food in the stomach remains alkaline for an appreciable length of time.

Diagnosis.—The diagnosis depends partly on the age of patient, partly on the information obtained by the passage of a bougie, and partly on any other evidence which may be obtainable of the existence of a tumour, and of the sequelæ to which it may have given rise. For example, there may be fragments of the tumour in the regurgitated matter, and a limited distention of the stomach, not due to aneurism or the pressure of a tumour without, renders the diagnosis of malignant stricture not certain. A small olive-headed probang, small enough to pass through the stricture once the initial spasm is relaxed, will, when the head is being withdrawn, give rise to a spasm again when it comes in contact with the lower end of the stricture, and by comparing the distances as indicated on a scale marked on the stem of the instrument, a definite measurement will be obtained of the vertical measurement of the stricture. Such information may strongly point in favour of malignant stricture. Further, blood is very likely to be present in the regurgitated matter in a case of malignant stricture, although it may also occur in aneurism.

The utmost caution should be practised before passing a bougie or probang, and aneurism must be excluded, because rupture might be induced and the death of the patient accelerated.

Prognosis.—It is extremely unlikely that a malignant stricture can be cured. Whatever the nature of the tumour, if it is malignant, it is a question rather of prolonging time the patient's life than of attempting any definite cure by operation or other means. Gastrostomy may add years to the patient's lifetime, and sometimes the passage of a bougie through the stricture, or the temporary introduction of a vulcanite tube, may cause dilatation of the stricture, and so permit of the passage of food with greater ease.

Treatment.—As already indicated, something may be attempted by the passage of a bougie, or by the introduction of a vulcanite tube, and, where these fail, by performing gastrostomy. The general treatment otherwise should consist largely in keeping up the patient's nutrition, and, when pain is present, administering opiates and antispasmodics such as ice, dilute hydrocyanic acid, and morphia.

(3) Dilatation and Diverticula

Dilatation is frequently secondary to and above the level of a stricture, and it may involve the whole of the wall of the œsophagus.

Diverticula are localised bulgings which may possibly be due to imperfect development, but are more likely to be the result of pressure from within, such as might be produced by a foreign body. The diverticulum is generally formed at the junction of the pharynx and œsophagus; it projects behind the gullet, and may bulge on both sides of the neck. It may be palpable when filled with food, and may produce dysphagia from the pressure of the pouch on the œsophagus. Traction diverticula occasionally develop. They are due to adhesion with glands, often at the tracheal bifurcation. The adhesions cause traction on the anterior œsophageal wall. The condition is amenable to operation, and surgical text-books should be consulted with reference to the procedure.

(4) Paralysis of the Œsophageal Muscles

This may occur in diphtheria and bulbar paralysis. It necessitates feeding the patient through an œsophageal tube, and the treatment, in diphtheritic cases at least, consists in electrical applications, the use of strychnine, etc.

(5) Rupture of the Œsophagus

A rare condition, most frequently found after death and due to post-mortem digestion. During life it may result from a tumour, or from the effect of a powerful corrosive on the œsophageal wall, but very exceptional cases of rupture as the result of strain have also been recorded.

VI. AFFECTIONS OF DIGESTION

(1) Normal Salivary and Peptic Digestion

THE ptyalin of the saliva acts in an alkaline medium, and salivary digestion continues in the stomach for about half an

after ingestion of a meal. By its action starch is converted into maltose and cane-sugar into glucose. Organic acids may be formed to some extent during salivary digestion. After about thirty minutes have elapsed, ptyalin digestion ceases, and hydrochloric acid and pepsin take its place. The gastric juice converts proteids into albumoses, and later into amino-acids; it curdles milk (rennin), and it acts in virtue of its hydrochloric acid as an antiseptic.

Free hydrochloric acid should normally reach a percentage of 0.5 per cent. It is true that organic acids can take the place of hydrochloric acid in the process of digestion with pepsin, but they do so in a very imperfect manner. Lactic acid is probably largely formed from butcher meat and bread. The flow of gastric juice is stimulated by the saliva. The peristaltic movements of the stomach greatly aid digestion. No change is effected by peptic digestion on carbohydrates or fats, but some of the products of salivary digestion may be absorbed by the stomach, such as maltose and grape-sugar, while salts and amino-acids are also taken up, the latter only to a very limited extent. Water is not absorbed by the stomach. Peptic digestion lasts for 5 to 7 hours, when the chyme passes on into the duodenum.

(2) Indigestion

There are many causes of indigestion, but the following unclassified list is suggestive:—

- . Huge meals.
- . Badly masticated food, whether from the habit of bolted food or from bad teeth.
- . Excessive dilution of the stomach contents, as for instance by drinking too much water, thereby rendering the gastric juice less efficacious.

Exercise too soon after meals.

Unsuitable food, which may be due to (a) a badly proportioned dietary, whether the excess be of carbohydrates or nitrogenous ingredients, (b) bad cooking, (c) certain indigestible articles of diet, such as crabs and lobsters, which have very long muscle fibres, and (d) excessive quantities of food.

Insufficient time between meals.

7. Interference with stomach movements owing to tight-lacing.

8. Excitement and emotion which may interfere with the process of digestion.

9. Existing pathological conditions constitute a numerous list. Bacteria in the mouth or about the teeth may interfere with both salivary and peptic digestion, anæmia and debility after any disease often predispose to indigestion, while local stomach affections, such as gastric ulcer, cancer, and gastrectasis or dilatation, are almost invariably associated with it.

10. Over-stimulation of the stomach by an excessive use of pepper, spices, pickles, and alcohol; closely associated as regards its detrimental effect is the consumption of undiluted spirits between meals.

VII. DISEASES OF THE STOMACH

(1) **Acute Gastric Catarrh; Acute Catarrhal Gastritis; Acute Dyspepsia**

AN acute catarrhal inflammation of the mucous membrane of the stomach.

Etiology.—(1) The ingestion of irritant, toxic, or decomposing substances into the stomach. This group includes, in addition to corrosive poisons, alcohol in excess.

(2) Unsuitable food or articles of diet, which although digestible in moderate quantities, in excessive amounts may give rise to gastric irritation. A good example of this is carbohydrates in excess, and especially sugar, or vegetable fibre, such as one ingests in eating salad, cabbage, and many other green vegetables. Certain muscular fibres, such as these in the flesh of crabs, lobsters, cockles, etc., being of great length, are extremely difficult to digest, and in excess may overtax the stomach.

(3) In rheumatism and gout, and in many persons who are said to have an "irritable stomach," there is a predisposition to acute gastric catarrh, and any indigestible meal may precipitate an attack.

(4) In many fevers with a cutaneous eruption, and especially scarlet fever and measles, the gastric mucosa may to a certain extent participate with the skin in the inflammation.

(5) In chronic Bright's disease there is frequently gastric tarrh, due doubtless to the presence of some toxic agent.

(6) In any organic disease of the stomach, whether simple, cancer, or tuberculosis, there may be acute gastric tarrh associated with the condition.

Pathological Anatomy.—The mucous membrane is reddened, there is increased mucous secretion, and a diminished flow of gastric juice. From Beaumont's observations on the stomach of Alexis St. Martin, it is certain that there are also small erosions and hæmorrhages. It is probable that the glandular epithelium, in place of secreting gastric juice, undergoes mucinoid degeneration. Should death occur, it is usual to find pigmentation present, possibly due to altered blood. There may be a definite change in the muscular coat, either hypertrophic or atrophic, while in the neighbourhood of the pylorus the mucosa may acquire a mammillated appearance, which is due to a small-cell infiltration around the orifices of the glands interfering seriously with their function. There is a great interference with the normal blood-supply to the stomach during digestion, and this fact, together with the structural alteration in the mucosa, will explain the diminution of both hydrochloric acid and pepsin, the former being usually more diminished in amount than the latter, although it may be completely absent in a very acute case. As a result, fermentative changes rapidly take place in the stomach contents, and organic acids are formed in great excess, adding much to the discomfort of the sufferer.

Clinical Features.—A very typical attack of gastric tarrh follows either an imprudent meal or a bout of drink-

There is generally malaise with epigastric discomfort, which may amount to severe pain, nausea, and vomiting. The tongue is thickly furred, there is loss of appetite, great thirst, and often a limited degree of pyrexia. The saliva may be increased in certain cases. The pulse is rapid, but soft, and in part of the malaise already described, there are generally shooting pains in the joints. Headache is a common symptom. In many cases of acute gastric catarrh in children, the inflammation extends downwards and causes diarrhoea, sometimes colicky pain, but in adults constipation is the rule. The inflammation of the duodenal mucosa, causing interference with the function of the ductus choledochus, implies that a degree of catarrhal

jaundice will follow the gastric catarrh in one or two days' time.

In many cases the condition might more properly be termed subacute than acute.

Diagnosis.—While the typical phenomena already described indicate an acute gastric catarrh, it is wise to ascertain whether some other condition may not also be present, and *typhoid fever* should be thought of and excluded by the Widal reaction, where there is any doubt as to the diagnosis.

Prognosis.—Generally in a few days the condition yields to treatment, but if too many attacks occur, a persistent chronic gastric catarrh will develop.

Treatment.—1. We should strive, in the first place, to get rid of any irritant present, such as unripe fruit, decomposing meat, etc. For this purpose an emetic, especially in the case of children, is of great value. Sometimes the stomach must be washed out either with a dilute alkali, such as sodium bicarbonate, or with a weak solution of permanganate of potash, this treatment being followed with a saline purge or a dose of Gregory's mixture. Should we suspect the presence of organisms, the internal administration of 10-15 grains of sodium hyposulphite will be advisable.

2. We must next endeavour to soothe the stomach, and in certain cases this should be done by giving it complete rest and feeding by the bowel. Hydrocyanic acid (℥ 3), bismuth subnitrate (gr. 10-20), oxalate of cerium (gr. 1-5), will be found of value to soothe pain, while opium and the use of ice, both internally and externally, are sometimes of advantage. It should be remembered that to certain patients the application of heat over the stomach is more helpful than the ice-bag.

3. Try to aid the stomach to return to its normal function by giving predigested food, and later hydrochloric acid and pepsin in proper proportions, once the irritation of the mucosa has disappeared.

(2) Acute Toxic Gastritis

A. This is a severe degree of the condition just described, and may be due to the swallowing of a corrosive poison. The inflammation set up is extremely acute, the vomited matter invariably contains blood, and from an examination of it an

ea may be obtained of the substance swallowed. The patient is usually in a state of collapse, and in many cases there is in addition marked inflammation of the kidneys.

Most of these cases rapidly prove fatal unless promptly relieved, and may eventually give trouble even when some antidote has been promptly administered, partly as the result of necrotic changes in the œsophagus and stomach, and partly as the result of the tendency to cicatrization causing stricture.

Sometimes emetics can be given, and if a suitable antidote obtainable the patient's life may be saved.

B. Another form of toxic gastritis is due to eating decomposing meat. This also causes a severe type of acute gastric catarrh, and may produce most intense inflammation. suppurative gastritis may ensue, dependent upon the nature of the organisms present, and the inflammation may spread to the intestine. Dreadful pain, vomiting, and diarrhœa, with rapid collapse, will follow, and death is only a matter of hours.

Even after a comparatively mild case, or where a very small amount of the decomposing food has been eaten, long protracted ill-health may result notwithstanding the most careful treatment.

(3) Phlegmonous Gastritis

In certain fevers, and especially diphtheria, there may be wide-spread suppurative inflammation involving the wall of the stomach, or one or more abscesses may form as the result of the presence of simple or malignant ulcers. In acute toxic gastritis there may also be suppurative inflammation in the stomach wall.

Dreadful pain, vomiting, and generally diarrhœa rapidly terminate a case which is absolutely incurable. Very rarely an abscess, by discharging into the stomach or some other way, leads to a favourable result, although such cases have been recorded.

(4) Chronic Gastric Catarrh; Chronic Dyspepsia

Chronic gastric irritation dependent on various causes.

Etiology.—Acute gastric catarrh may give rise in time

to chronic irritation of the stomach, and many of the causes mentioned already under the heading of acute gastric catarrh may induce a lesser but protracted degree of inflammation, and so bring about chronic gastric catarrh.

The causes of indigestion already described may also lead to organic change in the mucosa of the stomach, and as the result of backward pressure from the heart or lung there may be engorgement of the portal vein, and with it its gastric tributaries.

Pathological Anatomy.—There is frequently enlargement of the stomach and a grey tinge of mucous membrane (pigmentation), and the mucosa is covered with much tenacious mucus. Dilated veins in cases of backward pressure are not uncommon. There is sometimes thickening, sometimes dilatation with thinning of the wall of the stomach, and usually atrophy of the secreting glands; ulceration may be present.

Clinical Features.—There is generally tenderness on pressure over the epigastrium, and often discomfort after food, frequently nausea, and even vomiting; the other phenomena vary.

It is instructive to divide chronic gastric catarrh into several types; of these we take—

A. The Irritative Type.—This variety of dyspepsia is often due to the habit of overeating acquired in early life, or to the equally pernicious habit of bolting or insufficiently chewing food, while over-indulgence in alcohol, especially drunk undiluted, is a common cause.

The tongue is peculiarly diagnostic, being slightly furred on the dorsal aspect, red, and often somewhat fissured, and should all the fur be shed, it may resemble a raw beefsteak in appearance. There is great thirst, much uneasiness after food and discomfort between meals; the patient feels hungry, and then on beginning to eat is almost at once satisfied. He may have eructations, pyrosis, nausea, and even vomiting, and at the best he fidgets about until the discomfort becomes less marked. His temper is apt to be irritable, and his sleep heavy but unrefreshing, or he may suffer from insomnia.

Treatment.—Attention should at once be paid to the patient's diet, all heavy, salted, highly spiced, and smoked articles of food being eliminated. See that the patient eats slowly and at regular intervals, and attends, where it is

essary, to the condition of the teeth. Such treatment, properly carried out, and associated with a sufficient amount of exercise and care for the general health, may suffice to cure every case even without medicine.

Sometimes purgatives are necessary, and for a few days nuth and hydrocyanic acid may help in soothing the discomfort; occasionally the addition to each meal of hydrochloric acid (10 minims) with some form of pepsin should be given.

B. Oxaluric Type.—This type of dyspepsia is an unsatisfactory one, inasmuch as its exact pathology is uncertain, but we find that patients who are the subjects of this type pass large quantities of oxalates in the urine, and suffer certainly from a chemical error in digestion associated with inefficient digestive processes.

The leading clinical features of this form of dyspepsia are a pale, flabby tongue, generally marked by the teeth at the angles, a poor appetite, constipation, the presence of oxalates in the urine, and marked mental depression.

Treatment.—Regulate the diet, giving a proper mixture of vegetable and animal ingredients, try to increase the oxidative processes by enjoining walking or riding exercise, give occasionally some mineral acid such as dilute nitro-hydrochloric (M 10-20) or dilute nitric acid (M 10), and advise change of air and, if possible, a good and bracing holiday.

C. The Atonic Type.—Loss of nerve energy may imply peristalsis and also diminished glandular secretion. There may be merely stagnation of food in the stomach, or there may be actual retention with fermentation and subsequent vomiting.

Pathiology.—It usually results from debility following on severe illness, and especially fevers, anemia, and diseases of the nervous system, but physical and mental exhaustion predisposes. The exciting causes of the condition are improper diet, bolting of food, or anything which in the patient's debilitated state acts with detrimental effect.

Pathological Anatomy.—There is generally some dilatation of the stomach, often with thinning of the stomach wall. The gastric glands may be atrophied or merely temporarily acting abnormally.

Clinical Features.—A pale, flabby tongue, poor appetite, indigestion with some discomfort after meals, fermentative

changes, and sometimes diarrhoea, are typical. Headache is common. Where there is marked dilatation, with retention of fermenting stomach contents, the patient vomits sour stuff sometimes containing sarcinae.

The **Diagnosis** is usually simple, and the predisposing factor, such as a severe debilitating illness, helps. See "Gastric Dilatation" (p. 228) for the associated clinical features.

Prognosis.—Often very intractable, unless in a recent and not very severe case.

Treatment.—1. Aid the stomach by (*a*) supplying pepsin and hydrochloric acid to make up the deficiency; (*b*) increasing the gastric peristalsis by massage over the stomach, and sometimes by electrical treatment, general and local; and (*c*) ordering very simple diet.

In certain cases gradually increasing the work of the organ, by giving large quantities of milk and easily digested food, tones up the stomach more successfully than any other plan of treatment.

2. Improve the general health of the patient by administering general tonics, and by ordering change of scene and surroundings.

D. The Hepatic Type.—This variety is always associated with a sluggish liver, and is present in sallow, depressed-looking patients, who are aptly described by the word bilious. The tongue is furred and yellow, the eyes tinged with jaundice, and there is a bitter taste in the mouth. The appetite is uncertain, with no great hunger, but the patient, once started, may eat a fairly good meal. There is an uneasy sensation over the region of the liver, which may be slightly enlarged, and there is liability to attacks of catarrhal jaundice. There is marked constipation, and the motions are pale. The patient is fidgety and uneasy, and takes a gloomy view of life.

Treatment.—Give a cholagogue purge such as podophyllin (gr. $\frac{1}{4}$), euonymin (gr. 1-2), and combine with dilute nitrohydrochloric acid (m 10-15). Sometimes phosphate of soda, given in drachm doses thrice daily, greatly aids the process of cure. Remember the value of exercise in these cases,—walking, riding, golf, etc.—and write out a simple dietary, which should be given at regular hours.

(5) Nervous Dyspepsia

There are two distinct types.

TYPE A. Pain and discomfort associated with digestion, but normal capabilities of the stomach as regards both digestion and the quality and quantity of the gastric juice.

TYPE B. Great pain with digestion, with excessive secretion of gastric juice and sometimes vomiting. There may be great craving for food, or exactly the opposite. These cases are frequently obstinate. They are common in neurotic persons, and not infrequently the abdominal aorta pulsates violently.

Treatment.—Local blisters, combined with healthy surroundings and such tonics as strychnine and iron, are usually the best methods of treatment. Try valerian and other antispasmodic remedies. Avoid recommending alcohol and opium.

(6) Hæmatemesis ; Gastrorrhagia

Vomiting of blood from the stomach.

Etiology.—Ulcer of the stomach, simple or malignant, gastric aneurism, varicosity of the veins of the stomach, and congestion of the stomach wall, may all cause a varying degree of hæmorrhage. Simple ulcer of the stomach may lead to a very serious loss of blood owing to erosion of a considerable part of the stomach wall, and an abdominal aneurism might rupture through the stomach wall, death being due to the hæmorrhage.

In certain toxic conditions gastric hæmorrhage is common, for example, in some of the continued fevers, especially typhoid fever, and what may be termed the hæmorrhagic type of small-pox, typhus, scarlet fever, measles, etc.

Corrosive poisons cause severe gastric hæmorrhage, and it may also result from injury, such as might be produced by the excessive use of the stomach-pump.

In some constitutional diseases, such as hæmophilia, pernicious anaemia, scurvy, and other allied conditions, we may have hæmatemesis.

It may also occur from backward pressure from the portal system, as in common cirrhosis of the liver. Blood coming from the nose, mouth, or lung may be swallowed and then vomited.

Clinical Features.—The blood may either remain in the

stomach until partial digestion has occurred, when the vomited matter is dark in colour, resembling hare-soup or coffee-grounds, or it may be vomited bright red just as it is effused, in which case the hæmorrhage is more serious. Where bleeding has occurred some time before the blood is vomited, the patient may, coincidently with the hæmorrhage, feel sick and faint.

It should be remembered that if there is blood in the stomach, part of it will probably pass through the intestines, and that the stools will be of a deep black colour, similar to that resulting from the administration of iron: to blood in the stools the name *melæna* has been given.

The **Diagnosis** depends on the recognition of bleeding from the stomach as compared with hæmorrhage from the *lung*. In blood from the stomach it has generally a definite acid reaction; blood from the lung is alkaline. In blood from the stomach there is often partial digestion, whereas in hæmorrhage from the lung the blood is generally bright red in colour, unless it has been swallowed and then vomited. The blood in hæmoptysis is usually bright red and frothy, in other words, is mixed with air, whereas blood from the stomach may be clotted or fluid, but contains practically no air. The history of the case, the physical examination of the lungs and heart (especially mitral lesions), and the careful examination of the stomach ought to aid in the diagnosis.

The hæmorrhage from a simple ulcer is generally more copious and less frequent than hæmorrhage from a malignant ulcer of the stomach, and in addition the chemical investigation of the stomach contents often enables the physician to distinguish with fair accuracy between the two conditions.

Prognosis.—Excessive hæmorrhage is of course dangerous, and may lead to fatal syncope, while in other cases a limited hæmatemesis may be actually beneficial to the patient in relieving backward pressure, but the question of prognosis depends on the cause of the hæmorrhage.

Treatment.—The general treatment of hæmatemesis is to insist upon rest for the patient, and, above all, rest to the stomach. Food should be given by the bowel in severe cases, and only a little bit of ice to suck, or a few drops of iced water to relieve thirst, should be administered by the mouth. An ice-bag applied over the stomach may arrest hæmorrhage, while various remedies are of value, such as acetate of lead in

4 grain doses, ergotin in 1-3 grain doses, or hypodermic injections of ergotin or ergotinine.

An attempt should be made to soothe the patient by hypodermic injections of morphia, where excitement and nervousness demand interference. When it is safe to allow food by the mouth, it should be given in small quantities and often predigested. It is hardly necessary to add that the treatment of hæmatemesis in all its details must depend largely on the disease to which it is due.

(7) Erosions of the Gastric Mucosa

Hæmorrhagic erosions are extremely common in cases of backward pressure, whether from the portal system, the lung, the heart, and in many acute diseases similar erosions may be seen. They may bleed freely, and death has even occurred from the hæmorrhage. The remarkable cases of hæmorrhage following upon abdominal operation, and perhaps especially in cases in which a kidney has been incised, are often due to these hæmorrhages.

While these erosions may form gastric ulcers, and are in reality superficial ulcers, they should be described separately.

(8) Gastric or Peptic Ulcer, and Duodenal Ulcer

Perforating ulcer of the stomach.

Etiology.—Most common between the ages of twenty and thirty-five. Gastric ulcer is said to occur in about 5 per cent of all dead bodies, although not necessarily in an active condition, but this figure is apparently an exaggeration. Forty per cent are males, and 60 per cent of cases of gastric ulcer are males, and in men the age more nearly approaches middle life.

It is dependent on gastric digestion with, probably, a local vascular cause (possibly thrombosis or embolism).

Trades in which pressure is exerted over the stomach are said to predispose, as, for example, that of a shoemaker; and corset lacing in women is similarly prejudicial. Chlorosis and anaemia are often associated, and servant girls so affected are frequent victims.

Pathological Anatomy.—*Gastric Ulcer.*—Generally singly or 2 to 5 in number, usually situated on the posterior wall, near the pylorus, and specially near the lesser curvature.

They are rarely on the anterior wall, and when so placed are very dangerous, because they are apt to perforate without contracting any adhesions. The ulcers are round or oval, typically punched out, and generally terraced when chronic. This is due to the opening through the muscular coat being smaller than that through the mucosa, and that again smaller than the perforation through the peritoneum. They vary greatly in size, and may be very large, the larger ones being nearly always chronic. The ulcers may heal, become very chronic, break out again after healing, or they may perforate. Generally adhesions occur before the peritoneal coat is eroded, and the pancreas, left lobe of the liver, and omentum often contract such adhesions, while sometimes the ulcer opens into the lesser omental sac. The pancreas or liver may be deeply eroded, and large vessels, either in the stomach wall or in the organs just mentioned, may give rise to very severe or even fatal hæmorrhage, the pancreatic artery sometimes being opened into. Occasionally an aneurism forms in the floor of an ulcer. Following on perforation into the lesser omental sac there may be a sub-phrenic abscess, and possibly even subcutaneous emphysema; more rarely the ulcer perforates into intestine, pericardium, and elsewhere. Sometimes cicatricial contraction produces various changes in the stomach, as, for example, hour-glass contraction, but more frequently stenosis of the pylorus with secondary gastrectasis results.

Pathology.—The stomach wall is often more or less digested post-mortem, and slight erosions occur in acute gastric catarrh; if therefore the stomach wall is locally damaged, an ulcer may result from the action of the gastric juice. The terraced ulcer is believed to correspond to the area of blood-supply of an end-artery in the stomach wall. (1) Virchow thought that plugging such an artery with an embolus might be the explanation, while (2) Cohnheim contended that the diminished alkalinity of the blood in anæmia and chlorosis might permit the gastric juice to attack the stomach wall. (3) Gastric ulcer follows injury sometimes, but the theory of a preceding injury in every case is untenable, and no such theory, or for the matter of that no theory as yet formulated, seems to offer a satisfactory explanation. (4) Bacterial necrosis has also been suggested by Martin, but the evidence is insufficient.

Duodenal ulcers occur in the first part of the duodenum; they are more common in men, and they have been known to follow superficial burns over the region of the abdomen.

Clinical Features.—There may be no symptoms, or there may be more or less long-standing dyspepsia of varying degree. Bloating or aching pain is, however, common either in the epigastrium, where it is increased by pressure, or about the 11th dorsal spine or right shoulder blade. It is often relieved by altering the position of the patient so as to prevent the gastric contents from resting on it, and it is generally increased after a meal. In some cases, curiously enough, pressure relieves the pain. The pain is probably due to the action of the gastric juice, which is stated by Riegel, Ewald, and others to contain a great excess of hydrochloric acid. Vomiting may be induced in the sufferer, to relieve the excessive discomfort. Nausea and vomiting are common, and hæmatemesis is a prominent symptom. Giddiness indicates hæmorrhage into the stomach, and soon after partially digested blood is vomited, producing a typical hare-soup or coffee-ground-like vomit. If a large artery has been eroded, bright arterial blood may be brought

Blood is often passed in the stool, giving it the tarry appearance called melæna. Where hæmorrhage is very slight there may be no hæmatemesis, and only melæna may be present. Care must be taken not to confuse melæna with stools coloured black by bismuth or iron sulphides. Palpation of the epigastrium may not merely induce or increase the pain, but may yield definite evidence of a pyloric tumour largely due to thickening and cicatricial adhesions of an old ulcer.

Perforation may lead to peritonitis, in the first instance limited to the lesser omental sac, or to a general peritonitis, and perforation into the pancreas or liver may give rise to special symptoms and often severe hæmorrhage.

The clinical features of duodenal ulcer closely correspond to the account just given. Pain may follow the ingestion of food, but after a longer interval and when the gastric contents have passed on into the duodenum.

The **Diagnosis** may be difficult from want of symptoms, but the presence of pain following immediately on the ingestion of food, and associated with an excess and not a diminution or absence of hydrochloric acid, should make one suspect the presence of a simple gastric ulcer. Blood in the vomited

matter is very important. It is hardly possible for any length of time to mistake a *malignant ulcer* for a simple one, while *gall-stones* frequently cause not merely characteristic pain, but the occurrence of subsequent jaundice, and tenderness not so much over the stomach as over the liver.

When a gastric ulcer has ruptured, although general peritonitis and the typical symptoms of collapse taken with the previous history may render the diagnosis easy, there are cases in which much difficulty exists. The ulcer may rupture into the lesser omental sac, and it may be difficult to ascertain the cause of an abscess in that region. It is impossible, as a general rule, to diagnose with certainty the rupture of a gastric from a duodenal ulcer prior to operation.

The *gastric crises of locomotor ataxia* may occur without any ataxic or other cord phenomena, but these develop later, and the lightning pains, ataxia, and loss of knee-jerks rectify any mistake.

Prognosis.—In most cases it is good, especially if the patient be wisely treated, but the risk of perforation should not be forgotten, and in the event of its occurring prompt surgical treatment is necessary. It is possible that a long-standing simple ulcer may acquire a malignant character, although this seems to be more rare than we might reasonably expect.

Treatment.—Absolute rest to the stomach is essential, and it is prudent to feed entirely by the bowel for several days, simply giving a small quantity of iced water by the mouth to allay the local discomfort. An ice-bag applied over the stomach is often of value, while bismuth subnitrate (gr. 10-30), dilute hydrocyanic acid (℥3), and similar remedies may be administered for the relief of pain.

Hæmorrhage is a most alarming symptom, and demands energetic treatment; and should ice and perhaps the administration of several hypodermic injections of ergotin not suffice, the aid of the surgeon must be sought, and the hæmorrhage arrested by surgical interference.

Occasionally, it must be remembered, hæmorrhage is from superficial erosions, and not from an ulcer at all.

When pain and hæmorrhage have ceased, and it is probable that the ulcer has healed, feeding by the mouth should be cautiously and gradually resumed, and for a long time the

ient should be enjoined to avoid all indigestible articles of food, to aid the stomach by careful mastication, and to attend carefully to the bowels.

The presence of an ulcer near the pylorus may cause picture with gastric dilatation, and much benefit may be gained from having gastro-enterostomy, or, better still, gastro-duodenostomy performed.

The treatment of duodenal ulcer demands the same care.

(9) Cancer of the Stomach

Etiology.—The stomach is a common site of primary cancer, and the pylorus is the most frequent part of the organ involved. More rarely the cardiac end of the stomach and middle of the viscus is affected.

The age of patients affected with cancer is usually after middle life, and in connection with the etiology of cancer in the stomach it is important to remember that a simple ulcer may become malignant, and that sometimes definite gastric cancer precedes the recognition of a tumour.

Sarcoma of the stomach is rarely primary, although secondary nodules may occur in the wall.

Pathological Anatomy.—The commonest varieties of cancer of the stomach are scirrhus, which constitutes fully 75 per cent of all cases, columnar, medullary, and colloid cancer. The last form is really a degeneration of a medullary cancer.

The pylorus is the usual site of disease, and the lesser curvature more frequently than the greater. It is common to find adhesions between neighbouring viscera as the result of cancerous growths, and in these nodules suppurative changes take place, causing eventually a general suppurative peritonitis in some instances.

The stomach is often greatly dilated where the tumour is near the pylorus, and the fungating growth may cause repeated and sometimes serious hæmorrhage. If the tumour is near the cardiac orifice of the stomach, the viscus is usually of small size, while a neoplasm involving the middle region of the stomach may cause marked hour-glass constriction of the

When dilatation occurs, the muscular coat of the stomach

becomes weak, and almost invariably the gastric secretion is seriously affected, and it is usually stated that the hydrochloric acid diminishes almost to the vanishing point, thus permitting fermentative changes to occur in the stomach contents.

Occasionally cancer of the stomach is secondary to primary disease of the breast or of some other region, while the lymphatic glands, liver, peritoneum, intestine, lung, and pleura may all be involved secondarily when a primary tumour is situated in the stomach.

Perforation is not uncommon; it may occur into the general peritoneal cavity, sometimes into the lesser omental sac, or into the intestine, pleura, lung, or pericardium.

Clinical Features.—The *general* symptoms include progressive cachexia, with loss of weight and strength, while anæmia is usually dependent partly on the amount of interference with nutrition and partly on the loss of blood, which may be a feature of the case. There may be some pyrexia.

The *special* symptoms include pain, frequently shooting through to the back, and often of a gnawing or dragging character. It is sometimes increased by the ingestion of food and by external pressure. According to Head, the skin area, referable to the stomach, and over which pain may be felt, is situated between the nipple and the umbilicus in front, and the 5th to the 12th dorsal vertebræ posteriorly. Severe dyspepsia is another prominent symptom, often with inability to take food, and very frequent vomiting. The vomited matter contains a minimum of hydrochloric acid, often none at all, and, if the stomach is dilated, the gastric contents ferment, and *sarcinæ* and other organisms are associated, while blood is usually present. Hæmorrhage is a frequent symptom; the blood may be coffee-ground-like, or of brighter colour, depending on whether it is partially digested or not. Hæmorrhage is certainly present in a large proportion of cases at some time or other, although the nature of the tumour and the degree of ulceration are important factors.

In examining the stomach contents careful microscopic investigation should reveal *sarcinæ*, yeast fungi, and many other bacteria and fungi, although we rarely find small fragments of the tumour in the vomited matter.

Physical Signs.—*Inspection.*—The presence of peristalsis

is probably the most definite and significant feature, which, however, is only noted in cases of dilated stomach. A visible tumour is present in about one-third of the cases, and it may be seen to descend with the diaphragm on inspiration. Osler suggests that distension of the stomach with carbonic acid gas in suitable cases may demonstrate the presence of a tumour, or, at all events, the existence of peristalsis. Sometimes secondary nodules appear in the neighbourhood of the umbilicus, sometimes in other parts of the peritoneum. *Palpation*.—Careful palpation reveals the existence of a tumour in a large proportion of cases, and the movement of the tumour with the diaphragm is often easily felt. It is a remarkable fact that not a few pyloric tumours are excessively mobile. In most cases of tumour in this region the mass is felt to be hard and nodular. *Percussion* demonstrates, especially if associated with auscultation, the outline of the stomach, and it may be again stated that a pyloric tumour is frequently associated with dilatation, while a tumour situated at the cardiac orifice causes atrophy of the organ. *Auscultation* may reveal gurgling of gas through the pylorus and, where present, the existence of peritoneal friction.

Complications.—The most frequent complications are certainly due to secondary growths, and the lymph glands, liver, peritoneum, etc., may all be involved. Perforation has already been mentioned, while only in a small number of cases does a painful illness terminate by gangrene.

Diagnosis.—The existence of a tumour in such a large percentage of cases renders the diagnosis easy. The absence of hydrochloric acid is of great value, especially when coupled with the age of the patient; *simple ulcer* is more common in young persons, and is associated with an excess of hydrochloric acid. In *pernicious anæmia* the examination of the blood is distinctive, because in most cases of tumour the anæmia is of secondary development, and shows none of the characteristics of the pernicious type. In doubtful cases surgical exploration should invariably be carried out if there is the remotest chance of benefit.

Prognosis.—It is rare to find patients with gastric cancer living over two years. The duration depends much on the interference with nutrition, but as a rule a period of some months is all that the patient can count upon, and occasionally an

acute case terminates in a few weeks. It should be remembered that pyloric tumours are sometimes operable before secondary growths occur.

Treatment.—The only treatment possible is palliative, except in cases where removal can be carried out. Give easily digested food, and wash out the stomach in cases of dilatation with very great care, using a soft œsophageal tube. Sometimes antiseptics have been given by the mouth, such as carbolic acid or creosote. In many cases morphia is requisite, and for the comfort of the patient it should be freely administered. Sometimes the performance of gastro-enterostomy prolongs the life of the patient for many months, and greatly relieves pain and discomfort, while in cases of tumour near the cardiac orifice of the stomach gastrostomy is imperative.

(10) Dilatation of the Stomach; Gastrectasis

An *acute* form is described, which is extremely rare, and which is dependent on over-distension of the stomach with solids or fluids.

The *chronic* or usual form is generally dependent on either (1) pyloric stenosis, often the result of gastric ulcer, simple or malignant, although sometimes due to simple thickening, or (2) loss of muscular tone, which is a common sequel to chronic gastric catarrh, general debility, anæmia, etc. (3) In addition to the two foregoing factors there is a third, namely, the distending force of the stomach contents, fluid, solid, or gaseous.

Pathological Anatomy.—In dilatation due to pyloric stricture, the stricture may be the result of an ulcer, simple or malignant, or may be due to adhesions between the pylorus and neighbouring organs; but whether the condition is due to pyloric stricture or weakening of the muscular wall of the stomach, there is great enlargement of the organ, which, in place of holding some 50 ounces, the capacity of the adult stomach in health, may hold 120 or more ounces. The shape of the organ varies considerably; the stomach may retain its usual form, the dimensions only being greater, or it may become U-shaped, the lesser curvature being enormously lengthened, and the greater curvature often reaching in a marked case to the symphysis pubis.

The stomach contents generally ferment as the result of the presence of abnormal vegetable and bacterial agents; there is usually a diminution in the normal constituents of the gastric juice, and in fact both the hydrochloric acid and the pepsin may be nearly absent. Butyric acid fermentation, due to the presence of the *B. butyricus*, lactic acid fermentation, the result of the *B. acidi lactis*, and acetic acid fermentation, due to the yeast fungus, may all be present, and sarcinae ventriculi are almost always found in the stomach contents. Gases of various kinds are generated, and sometimes it is said that if a match is applied the eructated gas may even ignite (marsh gas) when emanating from the mouth.

The **Clinical Features** are A *local*, and B *general* and *reflex*.

A. The *local symptoms* include epigastric discomfort, often with pain, and, if dilatation is considerable, the vomiting of large quantities (5 or 6 pints) of fermenting stomach contents at intervals of 1 or 2 days, the contents containing sarcinae and other organisms just referred to. Distressing eructations of ill smelling gas and dreadful factor of breath render the patient disagreeable to himself and to those about him. Peristaltic movements are often observable over the region of the organ, and are easily stimulated by the application of anything cold to the skin, and colicky pain may be associated. The appetite is poor and the thirst often great. The bowels are constipated, and the nutrition of the patient suffers greatly.

Physical Signs.—*Inspection* shows generally the peristaltic movements already referred to, and sometimes bulging of the dilated stomach. *Palpation* reveals marked splashing, which is easily elicited if the stomach is full. *Percussion* gives a fairly definite outline of the greater curvature of the stomach, but should be combined with *Auscultation*, when the loud stomach note will be at once detected by the ear applied to the stethoscope even although the transverse colon covers part of the organ.

Other methods of defining the stomach consist in the introduction of carbonic acid gas into the organ by the administration of 20 grains of citric acid and a similar quantity of bicarbonate of soda in two separate solutions, or air may be pumped into the organ through an œsophageal tube. Another method has been suggested, namely, emptying the stomach and then pouring into it a measured quantity of fluid, and estimating the

level of dulness which 20, 30, or 40 oz. of fluid reveal by percussion, the patient being examined standing. None of these methods, however, is as simple as combining percussion and auscultation, the accuracy of which depends on the fact that the stomach is never empty of air or gas; the investigation should be carried out with the patient in the prone position.

The fundus of the stomach may interfere with the heart, causing palpitation, which is relieved by eructations, and a functional albuminuria is sometimes associated with a marked degree of gastrectasis.

B. The *general and reflex phenomena* can be readily appreciated; they depend on the loss of nutrition, and also on the absorption of poisons from the fermenting stomach contents. They include emaciation, headache, and general discomfort, and tetany has been noted as a not uncommon result of marked gastric dilatation.

The **Diagnosis** depends rather on the discovery of the cause of the dilatation than on its recognition. Frequently a tumour at the pyloric orifice is palpable, and the degree of patency of the pylorus may be ascertained by measuring the amount of solids and fluids put into the stomach and the total amount vomited up, remembering the amount of saliva swallowed. In a case where no fluid contents pass through the pylorus, the total amount vomited will therefore probably exceed the amount taken in because of the added saliva.

Prognosis.—In mild cases rapid improvement occurs with treatment.

The **Treatment** consists largely in careful dieting, where necessary, washing out the stomach with an antiseptic solution, such as boracic acid (4 drachms to the pint), permanganate of potash (10 grains to the pint), hyposulphite of soda ($1\frac{1}{2}$ drachms to the pint), and other agents, and frequently following this operation by a limited and carefully selected meal. The preliminary use of a solution of bicarbonate of soda aids in dissolving the glairy mucus with which the stomach wall is coated. Massage over the region of the stomach is often of benefit, and general tonic treatment, consisting not merely in the use of iron and strychnine, but also in residence in bracing hill air, the use of cold shower baths, and the careful regulation of exercise, meals, and hours of sleep, should be

tried. An abdominal bandage is often helpful; the internal application of electricity, sometimes one pole being introduced into the stomach, is of value, and the high-frequency currents appear to be of distinct advantage in certain cases of this kind.

In cases of very marked dilatation, while the stomach should, in the first instance, be treated by lavage, it is often prudent to think whether gastro-enterostomy or gastro-duodenostomy should not be performed, or whether it is possible to remove the pyloric tumour which is causing the stricture. While rest to the stomach is certainly beneficial, it is unlikely that in a very marked case mere feeding by the bowel will do much towards curing the patient.

Where there is fermentation present, antiseptics may be given by the mouth, of which the best probably are hyposulphite of soda in 10-15 grain doses, creosote in 1 or 2 minim capsules, and small doses of carbolic acid.

The acute form of dilatation rarely admits of treatment, because too often it immediately terminates in death.

VIII. CONSTIPATION

RETENTION of the fecal contents of the bowels due to sluggish action or other causes.

Etiology.—(1) It may be the result of habit. Inattention to the calls of nature is often due to careless upbringing, and dates from childhood, but in many busy lives the rush to work in the morning is responsible for the omission. In certain cases the cause is a painful fissure at the anus or hæmorrhoids, which renders defæcation more or less torturing to the patient, and so tempts him to put off the evil day. There are many constipated persons, and especially women, who constantly use enemata, and in such individuals the deficient natural action is partly due to this habit.

(2) Constipation may be caused by diminished or altered secretion. Diminution may occur as the result of a drain otherwise, such as follows excessive sweating after violent exercise, or a great flow of urine, or a tendency to dropsical effusion into the tissues. A too dry diet favours constipation, a fact which should be remembered. The secretion is sometimes altered, as in cases where bile, one of nature's

purgatives, does not enter the intestine or does so in diminished amount.

(3) Constipation may be due to deficient peristalsis of the gut, and also to laxness of the abdominal wall. Peristalsis certainly is diminished where bile does not enter the intestine in sufficient quantity, as mentioned above, but more commonly a sedentary life is responsible. Active muscular exercise greatly aids normal peristalsis. Certain articles of diet are admirable peristaltic agents, such as oatmeal, brown bread, vegetables, and fruits of many kinds, and their omission in proper amount from the dietary is apt to induce constipation. The abdominal wall, if lax, does not support the intestines, and so constitutes another common cause of constipation. This is most likely to occur in women after many pregnancies, and also in stout persons who have become thin. But a weak abdominal wall is also not infrequently seen in neurasthenic and debilitated patients.

(4) Lastly, there are cases in which there is obstruction. This may be the result of pelvic tumours or of a stricture of the gut frequently malignant in nature, and not uncommonly situated at the sigmoid or one of the other flexures of the large intestine. In certain cases constipation may occur from displacement (enteroptosis), ballooning, or other alteration or malformation of a part of the intestine, and generally it is the sigmoid or rectum which suffers in this way. The pregnant uterus interferes with the normal action of the bowels, while the displacement or distension of the intestine is sometimes due to a long mesentery, or may be the direct result of long-standing constipation.

After severe attacks of enteritis or dysentery, spasm of part of the bowel and especially of the colon may be present, and leads to severe and troublesome constipation which is really a form of obstruction.

Pathological Anatomy.—While strictures and tumours may be causes of constipation, the changes in the bowel are a sequel to the condition; these include distension more or less marked and permanent, thinning of the wall, ulceration, especially in the caecum, and often associated with small pouchings of the wall, in which little scybalous masses are frequently found.

Clinical Features.—A huge number of persons suffer

from constipation, and they may even neglect artificial means of relief without appearing to be much the worse, while others are rendered miserable if the bowels fail to act, naturally or with the help of medicine, for a period of forty-eight hours. The chief results are discomfort, bad breath, and thickly furred tongue, flatulence, sometimes colic, general loss of energy and depression of spirits, headache, irritability, and sometimes giddiness. Great stress has been placed on the evil effects of absorption from the bowel, but whether they cause chlorosis or not, certainly constipation and flatulence are not infrequently the predisposing causes of appendicitis. There are also pressure results on sacral nerves, causing neuralgia, and on veins, causing hemorrhoids, and possibly even, in very extreme cases, oedema of the legs. A rectum blocked with hard faecal masses may give rise to great distension of the bowel with gas, and faecal impaction may be so great as to simulate peritonitis from the severity of the consequent colicky pains, while vomiting, eventually becoming faecal, may even commence if relief is not obtained. On abdominal palpation we can feel the hard faecal masses, mostly in the large intestine and caecum. Sometimes these masses are tunnelled, and diarrhoea due to irritation may actually coexist with constipation.

The **Diagnosis** is generally obtainable from the history alone, but a careful abdominal examination is invaluable, and often when in doubt as to a supposed tumour, the bowels are first cleared out so as to get rid of all faecal accumulations.

The **Prognosis** depends on the duration of the constipation and the amount of trouble the patient is willing to take so as to effect a cure.

Treatment. A glance at the etiology enables the reader to anticipate the treatment. Try to inculcate the importance of going to stool every morning after breakfast whether inclined so to do or not, and the desire to go may be aided by a few minutes' sharp walk, or in men by a morning pipe, while not infrequently much may be accomplished by dietetic measures. Recommend the patient to drink freely of weak tea or coffee at breakfast, to eat brown or whole-meal bread, oatcakes, and perhaps, best of all, to take porridge made of fairly coarse oatmeal. Fruit at breakfast is invaluable, and especially oranges, apples, figs, and prunes, while a tumblerful of cold or warm

water sipped whilst dressing is helpful. At other meals besides breakfast, study and revise, if necessary, the patient's dietary, introducing plenty of vegetables if an insufficient amount is being taken.

Exercise is important, whether walking, riding, or cycling.

A great deal of good can be done, where the abdominal wall is lax, by recommending an abdominal bandage, and by ordering daily abdominal massage. The rubbing should follow along the line of the colon so as to assist natural peristalsis, and wet compresses worn in the morning have a considerable effect in certain cases. Electricity applied to the abdominal wall is also good.

There are many drugs suitable for different types of cases, and probably cascara sagrada, either as fluid extract in 20-60 minim doses, or extract in pill or tabloid form (gr. 2-6), is one of the best purgatives. It acts specially on the large intestine, does not gripe in most cases, although it is a peristaltic agent, and the dose can be gradually reduced. A splendid pill consists of extract of nux vomica (gr. $\frac{1}{4}$), extract of belladonna (gr. $\frac{1}{2}$), and aloin (gr. 2), or compound rhubarb pill (gr. 2-4), and it may be taken at bedtime. These remedies tend to cure, although acting as purgatives, while, on the other hand, salts must too often be continued when once begun. Magnesium sulphate (3 1-4) in warm water just before breakfast, and sometimes combined with dilute sulphuric acid (m 20-30), acts well, and so do Carlsbad salts, Friedrichshall, Hunyadi Janos, Apenta, and many other aperient mineral waters. Salts clear out the intestine, and are, therefore, useful when beginning treatment in cases where the bowel is overloaded, but it is well to return as soon as possible to the type of remedies first mentioned.

Enemata of soap and water, sometimes preceded by an injection of olive oil, and small enemata of glycerine (3 1-2), are often helpful, and especially are big enemata beneficial where there is any faecal impaction. Inject 60-100 ounces of an enema made of Castile soap dissolved in water about blood-heat, and precede, where necessary, with 10-20 ounces of warmed olive oil, which the patient should try to retain as long as possible. Place the patient on his left side with the hips raised on a pillow before administering the enema, and give the soap and water very slowly. Enemata are apt to

cause a habit, and they should not be used unless absolutely necessary. Sulphur and cream of tartar in equal parts, 1-2 teaspoonfuls of the mixture being taken at bedtime in a little water, is an old-fashioned but serviceable purgative, and amongst others which might be mentioned are castor oil, purgen, elaterium, compound jalap powder, and rhubarb in the well-known formula of Gregory's mixture.

Constipation in Infants

The causes of this condition are faulty diet, want of tone as regards the intestinal walls, and perhaps a deficient flow of bile. Congenital strictures occasionally occur, and may call for operative interference, while sometimes there is only a trifling band or adhesion. Intussusception and obstruction from the development of a kink or constriction by adhesions cause constipation, but they are treated of under intestinal obstruction. Sucklings depend partly on the purgative effects of their mother's milk, and attention has to be paid so as to secure a suitable maternal dietary. A dose of castor oil taken by the mother operates through the milk on the child, and it is well to remember the ease with which constipation can be treated in the suckling by this means. A soap suppository is a well-known nursery remedy.

Imperforate anus, for which operative interference is the sole chance of relief, belongs to the domain of surgery.

IX. COLIC

(ENTERALGIA)

IRREGULAR and spasmodic contractions of the intestine which give rise to pain. Renal and biliary colic will be described elsewhere. It is probable that in some cases of enteralgia the pain may be neuralgic, and not due to irregular peristalsis, but we have little definite evidence of this.

Enteralgia is a symptom of many different diseases; but it is a most important one, and as such it should have a separate description.

Etiology.—(1) It may be due to something decomposing,

irritating, or indigestible in the contents of the bowel, and also to drugs acting as powerful peristaltic agents in large doses (colocynth and jalap). Food suitable for a person of strong digestion may cause colic in another, and food imperfectly digested in the stomach may become an irritant because it ferments. Chyle may undergo normal decomposition too far up the intestine, and so lead to colicky pain. Lastly, faecal masses in constipation set up enteralgia, the masses stimulating irregular peristalsis.

(2) Disease of the bowel of many kinds causes colic—thus enteritis, cancerous stricture, intussusception, appendicitis, dysentery, and ulcerations set up severe colicky pain which is one of the chief symptoms present.

(3) There are causes of colic which may be termed nervous; probably lead colic is partly produced in this way, and nervousness and cold in susceptible persons may set up severe attacks of colic. Strychnine in large doses, and other toxic agents with a similar action, produce violent colic.

(4) Colic may be due in women to pelvic irritation, and is, in such cases, of reflex origin.

Clinical Features.—There are many varieties of colic, from a slight aching pain in the abdomen to a severe paroxysm which is almost unbearable, but it varies in intensity from time to time during an attack. The colic described here is not the colic which results from complete obstruction. The pain may be due to spasms in the large or small intestine, and it is often localised near the umbilicus, but may be in any part of the abdomen. It is relieved by pressure with the hand where there is little distension with gas, but it is greatly increased if associated with the presence of much gas. The pain may follow the passage of the food into the intestine, being due to what is called intestinal dyspepsia, or it may occur just before the bowels act, and may be relieved by such action. Often the passage of a little flatus per rectum is followed by disappearance of the pain. Diarrhoea may follow an attack of colic, and thus the removal of the irritant is secured. In cases in which the colic is due to obstruction, visible peristaltic contractions of the intestine are often seen through the abdominal wall.

The patient's face in a fairly severe case is grey and anxious, his hands and feet are cold and clammy, and drops of perspira-

on may be seen on his forehead. His pulse is small and of low tension, but generally there is little or no pyrexia unless inflammation is present. The abdomen is rigid, and often the costicles are retracted, while frequency of micturition is common. Vomiting is occasionally present, but its presence should make the physician suspicious of a serious cause for the colic.

The **Diagnosis** depends on the recognition of the cause of the pain, and chiefly whether it is of trivial origin or points to a serious condition, calling for prompt operative interference. In a trivial colic the pain is more spasmodic and less constant than in obstruction. Pressure generally relieves colic, where there is little distension with gas, while in obstruction it always aggravates the pain. On examining the abdomen less rigidity is found with ordinary colic than with obstruction, while the absence of vomiting and temperature generally points to colic, and the history of the case should be suggestive. The pulse may be helpful—small and feeble in colic, is rapid and often wiry in obstruction. Lastly, a purge eventually relieves colic, but intensifies the patient's suffering in obstruction.

The **Prognosis** is favourable in most cases.

The **Treatment** of a case of ordinary colic consists in clearing out the irritant, and giving a sedative at the same time. A full dose of castor oil, with 15 to 30 minims of camphor, is most efficacious, and should be combined with fomentations, a linseed poultice, or, in mild cases, an Epsom salt hot-water bottle applied to the abdomen. Salol, in doses of 20 to 40 grains, acts slowly but certainly, diminishing the fermentation and consequent distension of the intestine, and wood charcoal (gr. 10-20) may be given in cachets for the same purpose. In many cases an enema of 20-40 ounces of soap and water affords very rapid relief, and may be combined with the purgative and antiseptic internal treatment. In cases of very severe pain, opium by the mouth, or hypodermically, and an enema are most successful. Where a very severe irritant is in the intestine, a course of sedatives is necessary, but the chief point is to ascertain the cause of the pain, and then endeavour directly to treat it. Intestinal worms not infrequently produce much colicky pain, and the measures for their removal are stated elsewhere. Sometimes

dietetic treatment is requisite, but the relationship of the pain to the passage of food through the pylorus gives a confirmative hint in these cases. Hysteria may be responsible for pain truly colicky in nature, or merely simulating it, and valerian or other anti-hysterical remedy is often of value in such cases.

X. DIARRHŒA

1. In Adults

THIS is a symptom of many diseases, and consists in excessive discharge from the bowels of too fluid fæces.

Etiology.—Diarrhœa may be due to—(1) a general condition, and occurs in such diseases as pyæmia, septicæmia, etc.; or (2), and more commonly, to local changes, such as intestinal catarrh, ulceration, and the presence of irritants in the intestine.

There are three modes in which diarrhœa may be produced: (1) By increased peristalsis of the gut, as from the use of colocynth and similar drugs. (2) By increased outflow of fluid into the bowel from the mucosa, as from the administration of a dose of salts. We should add here that a somewhat similar condition obtains when too much fluid is taken in by the mouth. (3) Possibly there is in some cases diminished absorption of fluid by the intestinal wall.

There are endless causes of diarrhœa which fall under the first of these heads, and they include enteritis of all kinds, ulceration of the intestines, the ingestion of irritants, and local irritation at the anus or in the rectum, besides waxy degeneration of the whole or part of the alimentary tract.

Clinical Features.—Many varieties or types of diarrhœa depend for their characteristics on the cause of the condition, and all of the more important are described elsewhere. It is only necessary to mention summer diarrhœa, and the forms of diarrhœa which are due to some organism, and which occur in epidemic form, often the result of contamination of drinking water with sewage. Dysentery, cholera, and similar diarrhœa-producing diseases call for no detailed reference. Apart altogether from these forms we should remember, however, that nervousness and sometimes chill may cause diarrhœa in

individuals who are otherwise healthy, and these do not fall under any special group. It is well known that to a nervous person the prospect of an examination, or an appearance in the witness-box, may induce a well-defined, if brief, attack of diarrhoea. There are also individuals with very sensitive intestines, to whom a trifling quantity of a green vegetable would prove an irritant.

The number of motions varies greatly, and in excessive diarrhoea, as in severe forms of dysentery, they may number over a hundred in a day. Where there is much diarrhoea the motions are pale, and almost free from bile-staining, as in the evacuations of an Asiatic cholera patient. There may be colic or there may be tenesmus, by which is meant straining after the evacuation has taken place. The amount of colic present varies with the cause of the condition, and the tenesmus is often associated with enteritis, or ulceration of the large intestine, and especially of the rectum.

Remember that diarrhoea may be in reality the result of constipation, and that sometimes the fecal masses are tunnelled, and a well-marked diarrhoea may develop from the irritation.

The **Prognosis** depends on the cause of the condition and the possibility of successful treatment.

Treatment.—Try to remove the irritant, if there is one, and give a purgative, such as castor oil, when necessary. To check diarrhoea give astringents and antacids. Bismuth carbonate in 10-40 grain doses, chalk mixture, catechu, kino and ipecacuanha are invaluable. Opium is often necessary, and there are many excellent pharmacopœial preparations, such as the pulvis cretæ aromaticus cum opio (gr. 10-40), pulvis kino compositus (gr. 5-20), pulvis ipecacuanhæ compositus (gr. 5-15). Where ulceration is present, rectal injections of either silver nitrate, or starch and laudanum, are sometimes given, but much depends on the nature and site of the lesion.

By persons liable to diarrhoea from trivial causes a cholera belt should be worn, and a carefully revised dietary is absolutely essential, green vegetables and fresh fruits especially being limited to a very moderate amount.

2. Diarrhœa in Children

A child is more likely to suffer from dietetic diarrhœa than an adult. In the hand-fed infant improper diet is a common cause of looseness of the bowels (milk from a cow suffering from mastitis is a good example), while a chill, and many other causes, are apt to induce attacks. Epidemic summer diarrhœa is common, and is treated of separately. An infant suffers seriously from any prolonged drain on the system, and the more delicate the infant, the more rapidly does the strength run down. He becomes flabby, pale, and pasty looking; the face is pinched and the expression anxious. The tongue is furred, and the child is often fretful and whining, much of the discomfort being due to the presence of some degree of colic. There are different types of infantile diarrhœa depending on whether it is due to enteritis simple or tubercular, to cholera nostras or to cholera infantum, types more or less distinctive.

The **Prognosis** is only favourable if there is rapid improvement under treatment, dietetic or medicinal, as death is inevitable in a weakly child with protracted diarrhœa.

The **Treatment** varies with the nature of each case. Care should be taken to find out any error in diet, and in cases of fermentation, or where an organism is responsible, salol, charcoal, and similar remedies are of great value. For green stools in infants no medicinal agent appears to act so satisfactorily as dilute hydrochloric acid in 1-3 minim doses before meals. In many other cases a mild purge such as castor oil should be given, followed later by astringents. In place of castor oil the following powder might be substituted:—

R_x

| | | | | |
|--------------------------------|---|---|---|----------|
| Pulveris Rhei Radicis | . | . | . | gr. 1-1½ |
| Hydrargyri cum Creta | . | . | . | gr. 1-3 |
| Pulveris Ipecacuanhæ compositi | . | . | . | gr. 1-3 |
| Pulveris Cinnamomi compositi | . | . | . | gr. 1 |

Fiat pulv. ; tales 6.

Sig. : One powder to be taken at bedtime, and not repeated unless ordered.

XI. ENTERITIS

INFLAMMATION of the intestines in whole or in part. There may be a duodenitis, a colitis (colon), a typhilitis (caecum), an appendicitis, and a proctitis (rectum).

The following varieties of enteritis may be differentiated: (1) Catarrhal enteritis acute or chronic; (2) Diphtheritic or membranous enteritis, which generally implies a very intense inflammation with the formation of a membrane, such as may occur in diphtheria, although not necessarily due to the diphtheria bacillus; (3) Ulcerative enteritis, the ulcers being typhoid, tubercular, dysenteric, though sometimes simple in origin; and (4) Mucous colitis.

(1) **Catarrhal Enteritis in Adults**

Varies much in degree, and may be the result of many different causes, and it should be remembered that a very severe type of catarrhal enteritis may lead to the formation of a "diphtheritic" membrane, while ulceration may follow as a sequel.

Etiology. Irritants of all kinds, whether unripe fruit, decomposing meat, or arsenic, may set up enteritis. Excess of what, in smaller amount, would be wholesome, exposure to cold, and possibly even nervous causes, may all give rise to mild attacks. It is the experience of every physician that a mental shock, a period of great anxiety, and, perhaps most of all, excessive nervousness, can produce a catarrhal enteritis in susceptible individuals. But catarrhal enteritis may also be part of a specific fever, such as influenza, diphtheria, pneumonia, and typhoid, and it is even more commonly present in pyæmia and septicæmia. It may be a sequel to local inflammation such as an ulcer, tumour, or peritonitis. Lastly, in the terminal stages of many diseases, and especially Bright's disease, heart disease, common cirrhosis of the liver, leucocythæmia, Addison's disease, and cancer, a catarrhal enteritis may develop, probably due to an infection by organisms and allied in nature to pyæmia.

Pathological Anatomy.—Much depends on the stage of the condition, because mere hyperæmia disappears post-

mortem, and only a marked degree of congestion is visible after death. The valvulae conniventes always show very definite injection where the jejunum is seriously involved, and the tips are specially congested and covered with thick glairy mucus often blood-stained. Peyer's patches and the solitary glands are often swollen, and in certain cases the mucosa is infiltrated with pus and has begun to become shreddy. Very commonly small superficial ulcers are seen after death. In chronic cases there may be a varying amount of slate-grey pigmentation due to altered blood pigment, and the wall of the affected part of the intestine may either be thickened or thinned.

Clinical Features.—In most ordinary cases of acute catarrhal enteritis due to the ingestion of an irritant there is marked abdominal pain, colicky in character, and associated with, or following it, severe diarrhoea which ceases when the inflammation subsides, as it does soon after the irritating ingesta are got rid of. There is often flatulence, and there may be nausea and vomiting. Where it is more severe, blood is seen in the stools (melæna) with much mucus, while in very bad cases the inflammation may lead to paralysis of the wall of the bowel and obstruction with constipation. If the *small* intestine is involved there is very severe colic and a fair amount of distension, but little or no tenesmus, while the evacuations will almost certainly contain undigested food. On the other hand, if the *large* intestine is chiefly affected there is more tenesmus (especially if it is the lower part of the colon and rectum), more borborygmi or rumblings of gas, more mucus in the stools, but little undigested food, and it is said that the abdominal distension is apt to be greater. The appetite is much more interfered with by a catarrhal inflammation higher up the intestine. The motions are less and less bile-stained according to the severity of the diarrhoea. Thirst is always great, and the degree of pyrexia indicates fairly clearly the gravity of the case.

Where the bowel is paralysed and peritonitis is threatening, or has actually commenced, as in strangulated hernia, etc., there is acute obstruction with its typical clinical features, namely, a rigid, board-like abdomen and vomiting, rapidly becoming faecal in character.

The enteritis associated with chronic Bright's disease,

common cirrhosis of the liver, etc., is often very intractable, but although it rapidly kills the patient, there may be very little pain.

Lastly, a *duodenal* catarrh has distinctive features. It usually follows a gastric catarrh, and it is apt to cause catarrhal indigestion from the blocking of the ductus choledochus; in severe cases there is often much vomiting, but generally little diarrhoea, at least for a time: the appetite is lost, and should the patient drink so as to satisfy the intense thirst, the fluid is usually vomited.

Diagnosis. - Enough has been said to enable a diagnosis to be made between enteritis of the small and the large intestine. It should be remembered that an *appendicitis* may be part of a more extensive catarrhal colitis, but the pain is in that case limited to McBurney's point.

The **Prognosis** depends on the type of catarrhal enteritis present and the success of treatment.

The **Treatment** is dietetic as well as medicinal. Any cause, if still present, must be removed, and it is often well to begin with a dose of castor oil and laudanum similar to that recommended for colic. Rest in bed is essential, and either fomentations or linseed poultices should be applied to the abdomen or else the ice-bag. Bismuth subnitrate (gr. 10-30) and dilute hydrocyanic acid (℥3) is often insufficient, and opium must be given sometimes in the form of suppositories $\frac{1}{4}$ - $\frac{1}{2}$; sometimes hypodermically (gr. $\frac{1}{8}$ - $\frac{1}{4}$), or sometimes by the mouth. Starvation, with only little pieces of ice to suck, gives the inflamed intestine a chance of partial rest and recovery, and in cases of enteritis limited to the duodenum, even the small intestine, an attempt may be made to feed the bowel. Where food must be given by the mouth - and this is commonly the case - small quantities of milk and aerated water or concentrated beef-tea, chicken jelly, etc., should be administered, and either all given cold, and even iced, or else slightly warmed. On no consideration should hot and cold foods of food be partaken of by the patient during the same

Frequently chalk mixture, *pulvis cretæ aromaticus* cum (gr. 10-40), *pulvis ipecacuanhæ compositi* (gr. 5-15), and gentle mixtures containing the tinctures of kino, catechu, and kino (of each 5 $\frac{1}{2}$ -1), etc., are beneficial. Salol (gr. 10-30), charcoal (gr. 10-20 in cachets), and other intestinal anti-

septics are worth a trial in cases with much flatulence. Should diarrhœa continue, especially if associated with vomiting, it has been suggested that washing out stomach and bowel might be attempted, and in children this has proved successful. Once the patient has begun to improve, gradually increase the diet, adding fish cream or a similar easily digested semi-solid, and remember that it may be necessary to give partially digested food for a long time. The diarrhœa of chronic Bright's disease, common cirrhosis of the liver, etc., is very intractable, and all sorts of powerful astringents may have to be tried, such as the lead and opium pill (gr. 2-4).

Catarrhal Enteritis in Children

In young children the fault usually lies with the unsuitable food given, either sour milk or milk containing some bacterial agent, perhaps the most serious one being, not the tubercle bacillus, but the bacillus of Gaertner, which sets up an acute infective catarrhal enteritis. Many infants are fed on "what is going," which sooner or later in delicate children will cause enteritis, while others get an excess of some constituent of a normal dietary, such as milk sugar in condensed milk, to the exclusion of the fatty matters or cream of fresh milk. Catarrhal enteritis is more frequent in hot weather.

Pathological Anatomy.—Osler states that the normal stools of healthy, breast-fed children contain many organisms, but specially the *B. lactis aerogenes* and the *B. coli communis*, the others being more or less unimportant. In the diarrhœa of children the number and variety of potent organisms greatly increase. There may be not infrequently little follicular ulcers in the lower part of the small and upper part of the large intestine, related chiefly to the swollen solitary glands, but otherwise there is little to note.

Clinical Features.—In mild cases there are green stools, containing undigested milk and a varying amount of mucus, and the child suffers a little pain, and rapidly begins to fall below par, becoming flabby, pasty-faced, and emaciated, if not promptly treated. The sinking of the anterior fontanelle is a useful guide to an infant's general state of health. In more severe cases there is blood-stained mucus in the stools, which

very offensive; there is much pain and greater abdominal distension. Vomiting is common, there is frequently pyrexia, and the child rapidly dies, sometimes in convulsions, if not relieved.

There are also chronic cases in which the symptoms are no longer standing and less urgent.

Treatment.—Attention must be at once paid to the milk. If the child is breast-fed, it may be getting suckled too often, or the mother's milk may be poor. Sometimes a wet-nurse is desirable, but often milk, as closely resembling healthy mothers' milk as possible, should be substituted. A mild purge is beneficial to remove all irritating contents of the bowel, and precautions about feeding may be all that the case requires. See that the milk is not allowed to turn sour, and that milk cans and bottles are kept thoroughly clean. Raw meat juice is a valuable aid in bad cases, and after the preliminary purge, opium is often useful, while sometimes salol, β -naphthol, or wood charcoal controls fermentative changes. Remember that alcohol is necessary where exhaustion is extreme.

The following subvarieties demand a brief reference.

(a) **Cholera Nostras.**—This form of diarrhœa is probably due to a specific organism or organisms of which we are yet uncertain. It is specially common in children, although adults do not escape. It is spasmodic, and is often due to some fermenting ingesta. The patient is suddenly seized with vomiting and diarrhœa, and the diarrhœa may resemble Asiatic cholera in its character and severity, and the patient may rapidly sink and die in one or two days, often with convulsions. The case is closely allied to catarrhal enteritis. The treatment is troublesome, because the vomiting prevents the retention of the remedies. In severe cases hot bottles and, if collapse threatens, the intravenous injection of saline solution are the best kind of treatment. Where there is much temperature, cold water sponging, cold or even iced water enemata (with very great care), or the wet pack may be tried. In all cases sedatives and intestinal antiseptics should be administered.

b) **Cholera Infantum** is apparently cholera nostras in its character; at all events, it is indistinguishable from it, and it presents the same or rise to exactly similar clinical features.

(2) **Diphtheritic or Membranous Enteritis**

The term diphtheritic means that there is necrosis of the mucosa with, in addition, an inflammatory exudation similar to what occurs in diphtheria, although the name does not imply that Löffler's bacillus is in any way responsible for the membrane.

Etiology.—It is found as a terminal affection in chronic Bright's disease, pyæmia, and common cirrhosis of the liver, and in many continued fevers, such as pneumonia and typhoid, and it may result from certain poisons, and especially arsenic, mercury, and lead.

Pathological Anatomy.—There is often very intense inflammation, and all the coats of the bowel may be affected. The muscular coat may be infiltrated with pus, and the bowel gangrenous. In other cases the amount of membrane is very limited, and may only involve the glandular structures or the tips of the valvulae conniventes. The membrane is the result of a bacterial necrosis, probably in both the severe and the milder types.

The **Clinical Features** vary according to the severity of the inflammation. There may, in cases of extensive necrosis of the intestinal wall, be all the symptoms of acute obstruction; in less extreme cases, profuse diarrhoea, with the passage of much blood and mucus. The other symptoms differ; occasionally much pain is experienced, while in other cases it is absent. Vomiting is generally more or less severe.

The **Treatment** consists in an attempt to allay the local inflammation and to check the diarrhoea.

(3) **Ulcerative Enteritis**

This may be due to a great variety of conditions.

Etiology.—Tubercle, typhoid, dysentery, and cancer all cause special forms of ulceration, described elsewhere under their respective headings. Superficial ulcers are common, affecting especially the Peyer's patches and solitary glands in the different forms of enteritis, and commonly occurring in children. The ulcers may be superficial and limited, or more extensive, and resemble dysenteric ulceration, although it may not be due to the specific organism or organisms of dysentery. There are ulcers in the cæcum and colon which are due to hard scybalous masses, and the scybala may be found in small

vacuoli in the bowel, the mucosa of which is often ulcerated. Ulcers which do not appear to have any definite explanation may also develop in the large intestine. They are often single, and may resemble in appearance a gastric ulcer, and, lastly, ulcers may open from without into the bowel, as in a localised suppurative peritonitis.

The **Clinical Features** vary greatly with the nature of the ulcerative process, but diarrhoea is common in nearly all cases. The motions are often offensive, and contain pus, mucus, blood, and sometimes shreds of necrotic mucosa. Pain in greater or less degree is frequently present, and ulceration of the lower part of the large intestine is accompanied by tenesmus. The results of ulceration include perforation, pylephlebitis suppurativa, hypertrophy or atrophy, and sometimes constriction of the bowel.

The **Treatment** varies with the type present. Typhoid and dysenteric ulceration are described separately. Superficial ulceration should be treated on the lines laid down under bacillary enteritis in adults and children, and ulcers due to scybala are generally cured when the masses have been cleared out. For ulcers in the large intestines the reader is referred to the treatment suggested for dysentery. The one principle which must be remembered in treating nearly all intestinal ulcers is rest to the bowel, and a carefully studied diet, which should contain a minimum of waste matters. In ulcers situated in the small intestine, or even in the cæcum, an attempt may be made to feed by the rectum, provided, of course, there is little diarrhoea.

Tubercular ulceration may be primary (in children), but is generally secondary to pulmonary tuberculosis. The ulcers are chiefly situated in the ileum and colon. They affect the Peyer's patches and the solitary glands, and extend in the transverse axis of the bowel; if they heal, they readily induce stenosis. The edges are infiltrated, and the ulcers often involve the muscular coat, while tubercle nodules are generally seen in the corresponding peritoneum. Tubercular peritonitis, tabes mesenterica, and, eventually, waxy degeneration are commonly associated. The clinical features and treatment do not differ from the description given above, and, as a rule, the prognosis

(4) **Mucous Colitis**

This is a form of very chronic colitis, in which a large amount of mucus is passed by the patient, often as casts of the bowel.

Etiology.—It is commoner in women than in men, and is almost invariably associated with neurasthenics, who may be, and often are, very hypochondriacal.

The **Clinical Features** vary with the severity of the case. Generally attacks of mucous colitis develop from time to time, and the patient, after complaining of abdominal pain and discomfort, passes casts of the bowel, sometimes nearly a foot long, often only in bits, but always with a good deal of tenesmus. The colon is frequently distinctly palpable as a firm, cord-like structure which is tender to the touch. During the days of discomfort, while the pain lasts, there is much fretfulness of temper, and sometimes great depression of spirits.

The **Diagnosis** should be directed to the elimination of cancer, or other condition associated with much mucus in the stools.

The **Prognosis** depends on the patient's general health as much as on anything else. The disease may last for months or even years, and give rise to constantly recurring periods of discomfort.

The **Treatment** consists partly in dietetic measures and care for the general health of the sufferer, and partly in an attempt to treat the condition by remedies which probably to some extent exert a local action. Meals should be given at regular hours, and quantity and quality must be studied. The following medicinal remedies are good:—Arsenic and tar, sulphur and cream of tartar; half a drachm of the last two remedies may be taken together at bedtime. Washing out the colon has also been recommended, and is worth a trial, and castor oil is another valuable remedy. During the paroxysms of pain, hot applications may require to be applied to the abdomen.

XII. APPENDICITIS

THE appendix is about 3 inches in length; it is a blind tube, and its walls have the same structure as the cæcum. It

possesses lymphoid tissue and many mucous glands, and it has a distinct mesentery, in which run an artery and a vein. The appendix usually lies just under the lower end of the ileum, pointing towards the umbilicus, but it may be situated under the cæcum or hang over the pelvic brim, while it sometimes contracts adhesions with neighbouring structures. In 2 per cent of cases of appendicitis there is a concretion in the lumen of the tube, either faecal, or more rarely a grape-stone, cherry-stone, or other small foreign body. Sometimes the appendix is thickened, and the lumen is often partially constricted near its base or even obliterated, while a cystic distension of the tube beyond the constriction is usually associated. Ulceration is not uncommon, sometimes tubular or typhoid. Suppuration is frequent in the cystic end of the appendix, the most virulent organisms present being the *Bacillus coli communis*, the *Streptococcus pyogenes*, and the *Staphylococcus pyogenes aureus*. Perforation may rapidly occur in what are called fulminating cases, and as a result either the general peritoneal sac may be infected or a localised abscess develops intra- or sometimes extra-peritoneal in position. Occasionally an artery or vein is opened into, or phlebitis may be set up. Gangrene may also ensue either from the intense local inflammation or from interference with the blood-supply in the mesentery of the appendix. Generally an attack of appendicitis subsides without any of these complications arising, but it leaves some evidence of its presence behind, and recurring attacks are common; on the other hand, complete obliteration of the whole lumen would imply a natural and probably permanent cure.

Etiology.—The cause of appendicitis is still disputed. It certainly is in many cases due to constipation, although constipation alone is insufficient to produce it.

Clinical Features.—The anxious, flushed face and the hunched-up knees demonstrate the physical agony of the patient. The leading features are pain, sudden and colicky, generally in the right iliac region and increased by deep pressure over M'Burney's point. M'Burney's point is situated where a line joining the umbilicus and right anterior superior iliac spine is crossed by the outer edge of the right rectus abdominis muscle. There is marked immobility of the abdominal wall in the affected region. Between the attacks, or where the

condition is more chronic, the pain is dull and aching, but with the attack it becomes sharp and lancinating. The pain is possibly due in part to peristalsis of the appendix itself. Vomiting is an early feature, and is persistent during the attack. Any fluid drunk is generally returned at once, but only in very severe cases, amounting to acute obstruction, does the vomited matter become faecal. The temperature is raised (100°-102° F.), and the pulse, often soft and rapid, is a good index of the acuteness and gravity of an attack. On palpation a tumour-like mass is frequently felt, which is, often at least, due to the protecting abdominal muscles. When an attack is over, this resistance may disappear. There may be irritability of the bladder and sometimes albuminuria. The presence of an excess of indican in the urine has been noted. Where an abscess forms, a leucocytosis will probably develop, but a leucocyte count has not always yielded reliable results. An attack usually passes off in two or three days, leaving some aching pain behind.

Diagnosis.—*Renal* and *biliary colic*, *salpingitis*, *intussusception*, and *ordinary colic* can hardly be mistaken. Perhaps *intussusception* is most similar. Remember the red currant discharge from the bowel in the latter condition, and the fact that children usually suffer.

The pain localised to the right iliac region and the vomiting are practically constant symptoms of appendicitis, the former only disappearing, and that but temporarily, if gangrene ensues. *Perinephric* and *perityphlitic abscess* may simulate this affection, but *perityphlitis* is generally of appendicitic origin.

Prognosis.—Most cases recover without operation, and there may be no recurrence. Operative interference is good either within six hours of the commencement of an attack, or between attacks, and in every case of abscess formation operation is the only hope of cure. The death-rate should and would be low for operation cases, were medical men willing to get surgical aid promptly. Where general peritonitis and gangrene are present, the prognosis is bad.

Treatment.—Ice locally, morphia hypodermically, and enemata of soap and water constitute the best medical treatment.

Surgical interference is generally desirable, especially in

fulminating cases, and the death-rate for operation in quiescent cases is extremely small, and, in fact, should be *nil*.

XIII. INTESTINAL OBSTRUCTION

As the name implies, this is interference with the lumen of the bowel, and it is usually associated with complete constipation, although sometimes from the site of the obstruction the bowel may empty itself below the stricture, and thus mask the true nature of the condition present.

It may be acute or chronic—terms not always absolutely distinct, because a chronic obstruction may become acute; and under slightly different circumstances the same cause may act in one case by producing acute, in another case chronic obstruction.

Etiology.—The obstruction may be (1) Extramural, (2) Mural, and (3) Intramural.

1. *Extramural Causes.*—**A.** Strangulation of the bowel may be induced by bands or adhesions; by passing through apertures in the omentum, mesentery, or diaphragm; by passing into hernial sacs; by being caught by bands formed by persistence of fetal structures, such as Meckel's diverticulum, which is often connected with the umbilicus; by compression by tumours; and by peritoneal adhesions as the result of an appendicectomy or some other abdominal operation in which the peritoneum has been exposed. The cause of the strangulation may be actual compression of the bowel or interference with the blood-supply to the part. This somewhat complex group includes a big percentage of all cases of acute obstruction, probably approximating 40 per cent.

B. Volvulus, twist, or kink is another cause, and is most generally met with at the sigmoid flexure, although twists may occur elsewhere.

C. Intussusception, or invagination of one part of the bowel into another, is not uncommon in children. It occurs most frequently at or near the ileo-cæcal valve, that part of the small intestine being invaginated into the cæcum. The invagination rapidly increases until the mesenteric blood-supply of the bowel is interfered with, and intense congestion and

inflammatory œdema result, and if this is not relieved, gangrene will ensue. The invaginated bowel has even been known to reach the rectum, and to be palpable with the examining finger, but this would imply that the colon was invaginated into the colon or rectum. Occasionally sloughing occurs, and the invaginated part may be passed by the rectum, when natural cure might conceivably result, but this is very rare. In a case of the author's, a tumour at the ileo-cæcal valve, and primarily of the ileum, formed the starting-point and exciting cause of the intussusception. A classic sign of intussusception is the presence of a red currant jelly-like discharge per rectum. It should be remembered that in children especially, but sometimes in adults, invaginations are found post-mortem which are easily reduced with the fingers, and which probably occurred *in articulo mortis*.

2. *Mural Causes*.—**A.** One important group of these is apt to be overlooked, but is none the less a common form of acute obstruction. It is the paralysis of the gut, which inevitably occurs as a result of very intense inflammation, whether there is actual suppurative infiltration of the wall of the gut or not. In appendicitis the obstruction is often due to this cause.

B. Strictures of the gut or tumours of the wall of the gut.—These are more common causes of chronic obstruction, although at some stage in the affection the obstruction may become acute. They include rare congenital strictures which call for no detailed description, and cicatricial contraction of the bowel secondary to ulceration, especially tubercular and syphilitic, and much more infrequently typhoid and dysenteric. The tumours are sometimes simple, and include papillomata, adenomata, fibromata, and lipomata, but far more commonly they are malignant and are chiefly carcinomata. Carcinomatous tumours are generally situated at the sigmoid flexure, although they may be found at the hepatic or splenic flexures or elsewhere. Tumours of this description occur as a rule after middle life.

3. *Intramural Causes*. — **A.** There are many foreign bodies which may cause obstruction, and these include coins, fruit-stones, gall-stones, sharp-pointed objects such as pins, nails, hooks, and many indigestible substances which may accumulate until a large mass is formed. It is remarkable

how many large, pointed, or sharp objects, if accidentally swallowed, are readily passed per rectum, the best examples being pins, needles, and artificial teeth which, if not too large, may be evacuated without much delay. Should, however, a blockage occur from such an object, the cæcum is the favourite site, although it may be at any part of the intestine; in the case of gall-stones it is often in the ileum. Enteroliths or intestinal concretions are due to many causes. They may consist mainly of phosphate of lime and magnesia; they may be formed in part of husks of grain, of hair, or vegetable fibre, or they may be faecal masses impregnated with lime salts. Sometimes enteroliths are formed of some medicinal agent swallowed by the patient, such as salts of magnesium or bismuth. Enteroliths are, however, distinctly rare.

B. Faecal impaction is a frequent cause of obstruction, which is generally chronic, although it may become almost acute in the severity of pain and suffering to which it gives rise. A constipated habit, feeble muscular action of the intestines, and very probably badly masticated and unsuitable food, are amongst the commoner causes. An elongated or misplaced sigmoid greatly favours faecal accumulations. Above these more chronic obstructions the bowel is usually distended with much gas. Faecal masses should be definitely palpable, and therefore the condition is easily recognised; it is most common in women, and often occurs between the ages of twenty and thirty.

Clinical Features of Acute Obstruction.—The attack itself is sudden although there may be a history of chronic obstruction for some time previously. It sets in with acute and very severe abdominal pain, often felt at or near the umbilicus. The pain is at first colicky or spasmodic, then continuous, but with periods of greatly increased agony. Vomiting soon commences, first of stomach contents, then of bile, and later it is of a faecal character and is very persistent. There is generally absolute constipation, although the bowel may empty itself from the site of obstruction downwards, owing to peristalsis starting from the obstruction. The vomiting, it should be stated, is the result of reflex peristalsis starting from the site of the obstruction. Abdominal distension is not marked at first, but soon develops: it is due to gas, and the degree of such distension depends in part

on the site of the obstruction, being usually greater when the obstruction is situated low down in the intestine. With the distension, local tenderness to touch greatly increases, and the slightest pressure may cause intense agony. At this period the patient's appearance and attitude are very typical. His face is anxious and pinched, cold clammy sweat is seen on his brow, his tongue is covered with dry, brownish fur, and his thirst is intense. He drinks incessantly if permitted, but at once vomits up all he has taken. The urine is often greatly reduced in amount. The temperature is not necessarily above the normal, but the pulse becomes rapid and feeble, and death may result from collapse if he is not relieved within three or four days. There may be a definite leucocytosis. The clinical picture closely corresponds with that of acute peritonitis, and peritonitis may be found post-mortem.

Clinical Features of Chronic Obstruction.—There is no sudden onset, but rather a history of long-standing and very intractable constipation, with colicky pain from time to time and a good deal of general discomfort. There is frequently marked dilatation of the intestine above the obstruction, and visible peristaltic movements may be made out, and can often be induced by the application of a cold hand to the abdominal wall. There may be vomiting, but it is not typical of this kind of obstruction. There is practically always more or less abdominal distension, much increased if no movement of the bowels has occurred for some days. Sometimes attacks of diarrhoea supervene, occasionally the result of the constipation, in other cases due to irritation at the site of the obstruction, or directly due to the cause of the obstruction itself. The tongue is furred, and there is a good deal of general malaise, but no temperature. The nature of the obstruction can sometimes be recognised by palpation and percussion, and frequently most valuable information as to the part of bowel which is distended, and which is almost always above the stricture, is obtained by carefully combined percussion and auscultation so as to trace the continuity of the distended bowel. The method is described under the heading of gastric dilatation (p. 229). Much help may be obtained from examining the motions. In stricture near the anus the motions are often small in calibre or flattened, although a big prostate may be alone responsible for this phenomenon. Again, the presence of blood, mucus,

and shreds of tumour, etc., should be carefully noted. Not infrequently a chronic obstruction becomes acute, and then the symptoms mentioned under acute obstruction appear, and death rapidly follows if relief is not obtained: in any case the patient's health suffers from a comparatively slight chronic obstruction, and malnutrition, with its train of results, will ensue.

Diagnosis.—We must try to decide in a case of obstruction both where it is and what it is due to. Search first for hernia, and then most thoroughly examine the patient's abdomen by careful inspection, palpation, percussion, and auscultation, remembering the importance of any localised diminution in movement, or of the board-like resistance of the abdominal wall to the hand; and insist upon a careful rectal and, when necessary, a vaginal examination. Sometimes a ladder-like pattern of the intestinal coils, as described by Professor Wyllie, may be noted through the abdominal wall, but this is much more common in chronic than in acute cases. The site of the pain is always suggestive, and tenderness on palpation often localised. Much information is certainly obtained by one of the common methods of treatment, namely, the injection into the rectum of large quantities of fluid, and noting the amount which can be introduced. Careful percussion and auscultation before and after big injections of water teach a great deal, and sometimes the rectum has been inflated with carbonic acid gas (tartaric acid and bicarbonate of soda) for similar diagnostic purposes.

Often with information which is not too definite we have to proceed at once to operative interference, but whenever possible we should endeavour to find out beforehand the cause of the obstruction. In very young children intussusception is relatively common, and we can often feel the sausage-like tumour through the abdominal wall, while blood-stained mucus is very frequently passed per rectum (in quite $\frac{3}{4}$ ths of all cases). An appendicitis is recognised by the localised pain and the fairly typical clinical history of the case. Tumours and faecal obstructions are sometimes palpable.

The diagnosis between acute obstruction and other conditions is very important, but often very difficult.

(1) *Appendicitis* at a certain stage may lead to acute obstruction if not operated upon or relieved by treatment, but

to begin with it is not the same thing. In appendicitis there is the pain felt at M'Burney's point, while the vomiting very rarely becomes faecal—not indeed until the condition has actually become one of acute obstruction from paralysis of the bowel.

(2) *Hæmorrhagic pancreatitis* is very difficult to diagnose; the symptoms are the same as in acute obstruction, but the pain would probably, in the first instance, be referable to the region of the pancreas and the lesser omental sac. Local tender points on the abdomen are common, but the pancreas seems to be the chief site of pain.

(3) *Peritonitis* is often closely associated with acute obstruction, and when, as the result of peritonitis, the bowel is paralysed, it is in fact a form of acute obstruction, although there may be no actual narrowing, but rather a huge distension of the affected bowel. In peritonitis, however, the temperature is nearly always raised, the pain is much more continuous and less colic-like, and in general peritonitis the whole abdominal wall is board-like and motionless.

The **Prognosis** varies greatly with the nature of each case. An acute obstruction is always serious until relief is obtained by operation or other means, but many cases are necessarily fatal, and others are only saved by very prompt measures. The onset of gangrene is always grave, and death occurs in most cases. Chronic cases of obstruction may last for years, and even where a cancerous stricture is inoperable, as it too often is, the patient may live for months.

The **Treatment** must depend a good deal on the nature of the obstruction. In many acute cases the intervention of the surgeon is imperative, and that without delay. It is difficult without operation to reduce a strangulated hernia, and it is specially difficult to undo kinks and to rectify an intussusception. Inflating the bowel with air or fluid, best done while the patient is under an anæsthetic, has sometimes succeeded in cases of volvulus or intussusception; it is most likely to succeed in children, but too long delay is always dangerous, and surgical aid should be sought early. The awful nausea and vomiting is greatly relieved by washing out the stomach, and Kussmaul, to whom this most excellent suggestion is due, recommends it for its effect in reducing the painful abdominal distension. Pain should be kept in check by opium (morphia, gr. $\frac{1}{3}$ - $\frac{1}{4}$, hypodermically), often combined

with atropine (gr. $\frac{1}{100}$ – $\frac{1}{80}$), but it must not be forgotten that pain is a useful symptom, and its absence, artificially brought about, may cause a fatal delay in performing laparotomy. Certainly if enemata and inflation with air fail, they should not be too often repeated. From $\frac{1}{4}$ to $\frac{1}{2}$ of all cases of intussusception may be relieved by inflation, but it must be tried early—if delayed, inflation is more apt to cause rupture of the bowel, and much less likely to be successful. The death-rate after operation for acute obstruction is still very high, certainly well over 50 per cent, but then many cases are seen too late, and in a proportion of them no operation could do any good. Once the bowel is gangrenous, or a definite general peritonitis is developed, operation is not nearly so successful.

The enormous distension of the abdomen may be relieved by tapping with a fine needle through the abdominal wall, or by passing a long rectal tube up the bowel. Feeding the patient is usually a work of difficulty, and naturally we cannot in an acute case feed by the stomach. Small nutrient enemata are often advisable; merely give little bits of ice by the mouth to allay thirst and help to prevent vomiting.

In cases of chronic obstruction a great deal of benefit results from giving opium with atropine by the mouth or subcutaneously, and soon afterwards large soap and water enemata, which may need to be repeated. For faecal impaction faecal masses, if in the rectum, may require to be picked with a spoon; but in most cases they are situated too high, often in the caecum and colon, and then a preliminary enema of olive oil (\mathfrak{z} 6–12), combined in certain cases with an ounce of turpentine, should be administered, and about an hour after a big soap and water enema. This may have to be repeated several times before the patient is completely cured.

It is never prudent in chronic obstruction to give purgatives frequently; if one dose fails, it is preferable to resort to enemata. In certain cases where a tumour is present an artificial anus or a short circuit may be made so as to cut out the diseased part of the bowel.

XIV. INFARCTION OF BOWEL.

THIS is due to an embolus, most commonly from the heart in a case of ulcerative endocarditis, entering a branch of one of the mesenteric arteries. The result is gangrene of the loop of bowel affected. There are all the clinical features of acute obstruction with peritonitis, and the pain, vomiting, and rapid collapse are very marked. The bowel affected is found to be infiltrated with blood; there is no possibility of cure save by operative interference, at best a very slender hope.

Thrombosis of a mesenteric vein may cause the same results, although not quite so acutely.

XV. ENTEROPTOSIS

Glenard's Disease

DISPLACEMENT of various abdominal organs, often the result of lengthening of the mesenteric or other attachments. The kidneys most frequently suffer, and movable kidney and floating kidney are described separately. The liver and spleen are also sometimes displaced downwards, while the stomach, often as the result of tight lacing, may be considerably altered in position. Similarly, the colon may have a long mesentery, and sometimes intestinal obstruction is due to a kink occurring, owing to this long mesenteric attachment.

The **Clinical Features** vary considerably, but neurasthenia is not infrequently present in cases of enteroptosis.

The **Treatment** depends much on the organs involved and the degree of trouble which the displacement causes the patient. As noted elsewhere, the kidneys may be stitched back to the posterior abdominal wall, but it is more difficult by any abdominal operation to keep the liver or stomach in its proper position. Bandages and pads are often helpful, but the condition may cause much trouble to patient and physician alike.

XVI. DISEASES OF THE LIVER

(1) **Jaundice**

JAUNDICE used to be divided into, **A.** Obstructive or Hepatogenous, and **B.** Non-obstructive or Hæmatogenous; but it is doubtful whether all forms of jaundice are not really obstructive in origin, because, where a poison such as toluylendiamin is experimentally given, there is, it is believed, an increased pressure in the bile-ducts, due largely to an alteration in the viscosity of the bile itself.

A. Obstructive Jaundice, or Hepatogenous Jaundice

Etiology.—There are many causes of jaundice which can truly be styled obstructive, and the obstruction may be situated inside or outside the bile-ducts.

As examples of obstruction within the ducts may be mentioned gall-stones, cancer, foreign bodies, worms, inspissation of bile, and other less frequent causes; catarrhal inflammation of the bile-duct close to its exit into the duodenum is the cause of the mild attacks of jaundice in so many cases of gastroduodenal catarrh.

As examples of obstruction of the bile-ducts by pressure may be mentioned tumours of all kinds, peritoneal bands or adhesions and cicatrices, as in syphilitic cirrhosis, which may include the ductus choledochus, or one of its larger branches.

There are also cases in which jaundice has been induced by anger, mental shock, or emotional excitement, and it has been supposed that the obstruction of the bile-ducts is, in these cases, due to spasm.

The **Clinical Features** of an ordinary case of obstructive jaundice are described under catarrhal jaundice, but in addition to the transient tinging of skin and of urine by the bile, there are, in permanent cases, the very marked features which demand a word of description.

Permanent Jaundice.—The skin becomes dark greenish-brown in colour, sometimes almost approximating to black, the urine is deeply stained, the sweat more slightly.

The itching of the skin, often troublesome in catarrhal jaundice, is extremely irritating to the patient, and pruritus of a severe form may be induced, while other skin eruptions, such as nettle-rash and boils, occur.

In the urine there is nearly always albumin present as well as bile, while in the *Alimentary System* the stools in obstructive jaundice are absolutely colourless. Constipation, due to the loss of one of nature's best purgative agents, is the rule.

In the *Circulatory System* the slowing of the heart's action mentioned under catarrhal jaundice becomes very marked where the jaundice is permanent, and is probably due to the action of bile salts on the cardiac centres.

Petechial hæmorrhages are extremely common, and larger extravasations may also be present, while epistaxis occasionally occurs. There is often a difficulty in arresting hæmorrhage during surgical operations.

In the *Nervous System* there is, in the first instance, irritability, and later great mental depression. Eventually definite evidence of poisoning sets in, the patient becoming delirious, or suffering from convulsions.

The **Prognosis** is always unfavourable, unless it is possible by operative treatment to remove obstructions such as gall-stones.

B. Non-obstructive or Hæmatogenous Jaundice

This is always of toxæmic nature, and seems to depend on the rapid destruction of blood throwing a great excess of blood pigment into the liver, and thus causing an increase in the viscosity of the bile.

Etiology.—The common causes of this form of jaundice are (1) snake poisons, toluylendiamin, and the action of phosphorus and arsenic; (2) toxæmic jaundice may be present in certain fevers, and especially malarial fevers, yellow fever, sometimes scarlatina, typhus, etc.; and (3) toxæmic jaundice may occur in certain rare infective conditions, of which some are, and some are not, epidemic, and these include acute yellow atrophy, Weil's disease, and other kindred conditions.

The **Clinical Features** of toxæmic jaundice differ somewhat from those of obstructive jaundice. There may be less pigmenta-

tion of the skin and of the urine, and there is generally some bile in the stools. On the other hand, there are always much more marked evidences of toxæmia, with fever, delirium, sometimes convulsions, and often suppression of urine, while numerous petechial hæmorrhages may be noted.

(2) Catarrhal Jaundice

Jaundice due to the blockage of the orifice of the ductus choledochus is secondary to catarrh of the duodenum, which has extended from the stomach.

Etiology.—Gastro-duodenal catarrh may be secondary to many continued fevers, but is also a common result of any indiscretion in diet, and it is sometimes epidemic. Anger has been known to produce an attack. It may follow the passage of a gall-stone down the ductus choledochus, and it occurs in more chronic form in cases of backward pressure specially affecting the portal circulation.

The **Clinical Features** are those of obstructive jaundice, with typical clay-coloured stools; jaundiced skin, conjunctivæ, and soft palate; and with bile in the urine, following on an attack of gastric catarrh. Not infrequently there is no very definite history of gastro-duodenal disturbance, but close examination may elicit information as to some very indigestible article of diet which has been eaten. The skin may itch, and, it is thought, to the bile acids irritating peripheral nerve-endings, and on examination the abdomen is often somewhat swollen, the liver slightly enlarged and tender on firm palpation, and the gall-bladder distended with bile. The pulse is slow, falling to about two-thirds of the rate normal to the individual, and the patient feels languid, sleepy, and unfitted for any mental effort. Otherwise the symptoms depend on the gastric catarrh. The coloration gradually fades, and should completely disappear in a few days, although it may persist for one or three weeks. A longer duration, and the continued presence of bile in the stools, make the physician suspect either gall-stones or malignant disease blocking the common bile-ducts.

The **Diagnosis** depends on the recognition of the cause, rather than on the existence of the jaundice. A simple catarrhal jaundice should be unaccompanied by pain or emaciation, and

the jaundiced colour rarely becomes greenish, while petechial hæmorrhages are not common. *Weil's disease* should be kept in mind; its epidemic character is helpful, differing in this respect from most cases of catarrhal jaundice.

The **Prognosis** is favourable in a simple case, and recovery should not be delayed over a few weeks at the longest.

The **Treatment** is directed to allaying gastro-duodenal catarrh. Careful dieting, mostly milk, and sedatives, such as bismuth subnitrate (gr. 10-30), and dilute hydrocyanic acid (M 3) in mucilage and water are usually all that is necessary.

Rest in bed and warmth, with a saline purge and a small dose of such cholagogues as calomel (gr. 3-5), euonymin (gr. 2-3), or podophyllin (gr. $\frac{1}{4}$ - $\frac{1}{2}$), are beneficial, and sodium phosphate, given as a constituent of a saline draught, is one of the very best remedies. Fomentations, or the application of cold externally, are advisable where there is pain; in most instances the former is preferable.

(3) **Icterus Neonatorum**

In newly-born children there is commonly a mild degree of jaundice, which develops about the 2nd or 3rd day, when the urine may be bile-stained and the stools colourless. It is difficult to offer a satisfactory explanation. It is said to be due to a catarrhal jaundice, which seems unlikely. It may be due to diminished tension in the portal vessels, a not very comprehensible explanation, or to destruction of the enormous excess of red blood corpuscles found in the child's blood after birth.

A much more severe type may follow congenital stricture of the ductus choledochus, syphilitic cirrhosis, and septic poisoning from the stump of the umbilical cord. In this latter group of cases there is not infrequently hæmorrhage from the stump of the cord.

(4) **Acute Yellow Atrophy of the Liver** (**Icterus Gravis**)

A form of malignant jaundice in which there is rapid destruction of liver cells and diminution in size of the organ, and in which the chief clinical symptoms are cerebral.

Etiology.—A rare disease, most common in women between

20 and 40, and certainly closely related to pregnancy. It is possible that common cirrhosis of the liver has a causal connection with this disease. Organisms and bacilli have been described in several cases, but their rôle is not yet clearly made out. The disease is almost certainly of toxic origin, although whether or not due to a specific bacillus, situated locally, is a matter of doubt.

Pathological Anatomy.—The liver is small, often under 2 pounds in weight, and its capsule is wrinkled. It is soft and flabby, and it is of a dark green colour. The typical patches, some of them dark red, others bright yellow, are best seen on section. In the yellow or more recent parts, there is marked necrosis of the liver cells, many of which are reduced to an almost unrecognisable, granular-looking condition. In the orange or red patches there are in addition some secondary fibrous changes replacing the necrotic cells, the condition being really a cirrhosis, and the areas so affected have shrunk. Crystals of leucin and tyrosin may be seen in the liver, and may be obtained from the urine. Hæmorrhages are common, as they are in all forms of toxic jaundice, and are sometimes subcutaneous, sometimes subserous. There is marked bile-staining of the internal organs, and the spleen is generally enlarged, while the kidneys may show advanced catarrhal changes in the epithelium of the convoluted tubules.

Clinical Features.—The disease begins like an ordinary catarrhal jaundice with vomiting, but later head symptoms develop, sometimes convulsions, generally delirium, and these are followed eventually by coma. With these there is very deep jaundice, and subcutaneous hæmorrhages appear, while the urine is albuminous, of course bile-stained, and contains leucin and tyrosin, got in a majority of cases after evaporation. Leucin occurs in yellowish spherical crystals, with both faint radial and also concentric striation, and the tyrosin in needle-shaped crystals, which appear in sheaf-like bundles. There is not necessarily much pyrexia—possibly there may be none at all—the pulse is rapid and feeble, and the patient is obviously *extremis*. The spleen is found to be enlarged, and on diminution of the liver the percussion dulness anteriorly may be greatly reduced or almost absent.

As a rule the case rapidly goes downhill, and death follows about a week.

Diagnosis.—Too much stress must not be laid on finding leucin and tyrosin in the urine, because they may be present in other diseases, although rarely, and they may be absent in acute yellow atrophy. An ordinary *malignant or permanent jaundice* is always associated with head symptoms, but here they are specially severe, and if there be diminution of hepatic dulness with leucin and tyrosin in the urine, we need have no doubt of the diagnosis. *Phosphorus poisoning* most closely resembles acute yellow atrophy with its clinical features of sickness, diminution in liver dulness, and the presence of head symptoms, but there should be a history, and leucin and tyrosin are rarely present.

The **Prognosis** is very bad, although a few apparently authentic cases of the disease have recovered.

The **Treatment** is directed to the relief of symptoms, and to support the patient's strength.

(5) Cholelithiasis ; Gall-stones

Gall-stones are composed of (1) a nucleus which may be, and often is, inspissated bile pigment, or bacteria which have probably reached the gall-bladder from the duodenum, and which are *B. coli communis*, the typhoid bacillus, the pneumococcus, and other organisms; and (2) stratified layers of cholesterin and lime formed round the nucleus, although other salts may also be present in smaller proportions. Cholesterin and lime are found in excess in connection with inflammation of mucous membranes, and although cholesterin is usually retained in solution in bile even when in excess, the presence of one or more "nuclei" may lead to its deposition, and so, in time, to the formation of gall-stones. The glycocholate and taurocholate of sodium, present in bile, help to keep cholesterin in solution. Their deficiency may mean its precipitation, and this may be due in part to want of sufficient nitrogenous diet. Naunyn has conducted much research in connection with this subject, and has fairly definitely proved that fatty degeneration of cells in the mucosa may lead to excessive formation of cholesterin, and possibly a cholesterin which is specially apt to be precipitated. Where a part or the whole of the gall-bladder is absolutely shut off and contains no bile,

not only mucus, the gall-stones are almost pure cholesterin, whatever the nucleus may prove to be.

Etiology.—Gall-stones are most common in women. In the Ritter's collection of nearly 100 specimens more than three-quarters were obtained from women. They are certainly most frequent after middle life, and in women who have borne children. Marriage is held to be a predisposing factor, and this seems very probable. A sedentary life is certainly favourable to their development, and, in fact, anything as regards the habits of work of the patient which favours stagnation of bile in the gall-bladder or bile-ducts. Persons fed chiefly on carbohydrates appear to be more predisposed to gall-stones, which are less common in countries where meat is eaten as the staple diet. Perhaps their presence in gouty persons may be due to the limited meat dietary and the excess of carbohydrates. They are frequently met with in cases of cancer of the bile-ducts, though the question arises as to whether the gall-stones are always the result of the malignant stricture of the bile-ducts or not sometimes the cause of the tumour.

Gall-stones—their Characters.—They vary greatly in size from minute calculi, which are composed mostly of inspissated bile, and are soft and very numerous, to large stones which occur singly and may be several inches in length. When there are several stones in the gall-bladder which can rub against each other, they are faceted. Occasionally several stones occur without being faceted, but this is rare. The colour depends much on the amount of bile pigment, and large stones often contain a considerable proportion of lime salts. Stones may be found in the bile-ducts which perhaps have formed there, or have originated in the gall-bladder. Sometimes stones develop in the bile-ducts in the liver substance; these are usually small and ovoid, and mostly formed of bile pigment.

Clinical Features.—In many cases gall-stones are discovered after death which have never caused any symptoms during life, and the gall-bladder may even be full of gall-stones, forming a palpable tumour without producing one single attack of biliary colic. It is desirable, however, to describe a typical attack of biliary colic, and then to consider the other possible results of gall-stones.

Biliary colic is due to one or more gall-stones travelling from the gall-bladder along the cystic duct, and either passing down

the common bile-duct, passing back again into the gall-bladder, or becoming temporarily or permanently encysted somewhere. The pain is intense and agonising, and occurs in paroxysms, generally referred to the region of the gall-bladder, and radiating to the right shoulder, often accompanied by vomiting, profuse cold sweating and pallor, and generally followed by an attack of jaundice due to the catarrhal inflammation consequent on irritation caused by the stone. There is aching pain felt after the attack has subsided, and the gall-bladder may be distended and painful on pressure. A temperature of 102°, 103° F., or even higher, may be associated with the attack, but there is greater pyrexia if a cholecystitis develops. The presence of a definite attack of jaundice depends on whether the common bile-duct is involved in the journeyings of the stone, and will likely be absent when the cystic duct is alone affected. There is sometimes a transient albuminuria. It is difficult to offer a good reason for the onset of an attack of biliary colic. Probably exercise, a sudden strain, and perhaps indigestion may all aid in starting a gall-stone on its wanderings, but, on the other hand, attacks are not infrequently nocturnal. The motions should be inspected after an attack, in case the gall-stone or stones have been passed by the bowel. Sometimes many stones are passed, either in one motion or in successive motions. The appearance of a big stone in the stools implies that it has ulcerated into the bowel. Should the stone be impacted in the cystic duct, the gall-bladder may greatly enlarge from distension with mucus and may be readily palpated. If the stone is impacted in the common bile-duct, not merely is the gall-bladder enlarged, but the liver also becomes swollen and painful, the bile-ducts being distended with bile. Permanent jaundice may develop, or the stone may not completely obstruct the duct and may permit of the passage of some bile. Occasionally the gall-bladder becomes enormously enlarged, generally after blockage of the cystic duct, forming a huge abdominal tumour. In such a case the gall-bladder may contain mucus or even pus, with very little, or no, bile.

It should not be forgotten that biliary colic may be very severe, as just described, or it may be comparatively mild, or it may be almost unrecognised by the patient as an attack at all. Generally, however, one attack is the precursor of others, and a number of attacks should make the diagnosis quite clear.

If a stone is present in the liver, it causes more marked rigors.

The Results of Gall-stones.—One of the chief results is certainly suppuration. It may occur in the gall-bladder, causing a cholecystitis, or it may occur in the common bile-duct and its branches in the liver. In the gall-bladder there is risk of rupture into the peritoneum; in the bile-ducts, when suppuration ensues, there are rigors, enlargement of the spleen, and eventually the rapid development of pyæmic phenomena.

As already noted, impaction of a gall-stone in the common bile-duct may lead to permanent jaundice; in other cases the calculus or calculi may ulcerate into the intestine or elsewhere. Fistulous openings are sometimes met with into the duodenum, directly into the stomach, and sometimes into the peritoneal sac, while there may be communications with the portal vein or between the gall-bladder and the bile-ducts. Occasionally fistulous openings are external, and gall-stones may be discharged in this way.

Intestinal obstruction may be due to blockage of the bowel with gall-stones.

Abscess of the liver is a rare result of gall-stones, and it implies the presence of suppurative organisms, and in a cholecystitis the pus has been known to perforate into the liver substance and set up a liver abscess.

A form of cirrhosis of the liver is due to permanent obstruction of the bile-ducts, and hence gall-stones may, though rarely, be responsible for this condition.

We have left until the last the important bearing of gall-stones on cancer. Almost certainly cancer of bile-ducts is in many cases due to the irritation of gall-stones, although stasis of bile, the result of a cancerous stricture, may also lead to the formation of calculi. The gall-bladder is not an uncommon site for primary carcinoma, and in about 95 per cent of these cases gall-stones are present.

Diagnosis.—There are a number of conditions in which abdominal pain occurs, but none are easily confused with biliary colic. In biliary colic the pain is local, shoots typically to the right shoulder, is accompanied by vomiting, and is associated with tenderness over the gall-bladder, which is usually distended and palpable. In *renal colic* the pain shoots down to the testicle in males, there is no palpable gall-

bladder, and blood often appears in the urine. In *appendicitis* the pain is felt at M'Burney's point, and not over the gall-bladder, and there is generally constipation. The use of the X-rays should not be forgotten as a possible means of recognising gall-stones.

The **Prognosis** depends much on circumstances. An impacted gall-stone may be grave if the condition necessitates a serious surgical operation. Suppuration renders a case more anxious, and any suspicion of cancer is, of course, bad. Operation is much more hopeful than it used to be, and one attack of biliary colic generally means that other attacks are probable unless something radical can be done.

Treatment.—For the attack of colic at once apply fomentations or linseed poultices, or order the patient to take a hot bath, and aid in relaxing the spasm by giving morphia (gr. $\frac{1}{8}$ - $\frac{1}{2}$) hypodermically, or resort to general chloroform anæsthesia. Giving the patient large draughts of warm water to drink is also useful, although they may very frequently be vomited.

Between the attacks the treatment is much less clear and defined. Can we dissolve the calculi present in the gall-bladder if any are there, and if not, should an operation be performed? Gall-stones certainly are the result of stasis of bile, and therefore a free flow of bile should be encouraged. Sodium phosphate, in drachm doses thrice daily, is found of great service in many cases, and nearly all forms of purgative saline waters are good. Gall-stones are, to a certain extent, soluble in oil, and olive oil (or salad oil) is sometimes taken by the mouth in the hope that, somehow or other, it may help in dissolving the gall-stones. Patients do improve occasionally under this treatment, but probably not on account of it alone. Abdominal massage over the liver has often been recommended. Many spas are of value, and especially Carlsbad, which has a great reputation for the treatment of gall-stones. Vichy and Neuenahr are also much favoured. Plenty of exercise and a careful dietary are necessary for individuals who are subject to biliary calculi.

There is no special danger in performing cholecystotomy nowadays, further than the risks accompanying any abdominal operation, so that if no relief follows medical treatment, and there is no doubt about the existence of gall-stones, the operation can be recommended with confidence. Stones in the

cystic duct are easily removed as well as those which may be in the gall-bladder. Where there is suppuration (cholecystitis), operation and draining the gall-bladder is necessary.

Where the stone is in the common bile-duct, it may be crushed between the fingers or between the blades of a pair of forceps guarded with gauze, or it may be needled, or, lastly, it may be removed by incising the duct and stitching up the opening made.

Should it be found impossible to remove the obstruction from the cystic duct, the gall-bladder should be connected with the bowel (cholecyst-enterostomy). Where there is a calculus in the liver, it is often more difficult to reach, and the operation proportionately more dangerous.

If the patient suffers much from the itching of jaundice, he may sponge the skin with a solution of chloral hydrate (ʒ 4) and liquor ammonii acetatis (ʒ 2) in 10 ounces of water, or he may use a dusting powder of camphor (ʒ 1½), oxide of zinc (ʒ ½), and starch (ʒ 1), as recommended by McCall Anderson.

(6) **Active Congestion or Hyperæmia of the Liver**

In a slight degree it is physiological, and is the result of an indigestible meal, while in more marked degree it follows the ingestion of too much food, irritating articles of food, and especially alcohol in excess. In alcoholic subjects pain over the liver is very suggestive. In tropical countries, and probably as a part of many continued fevers, especially in tropical countries, there is apt to be hepatic hyperæmia. Where there is a sudden arrest of hæmorrhage, as in profuse menstruation in patients suffering from hæmorrhoids, pain over the liver is thought by many authorities to indicate hyperæmia of that organ.

The **Treatment** consists in a careful and simple dietary, keeping the bowels open, and complete abstinence from alcohol.

(7) **Passive Congestion or Passive Hyperæmia of the Liver**

This is the result of backward pressure in the hepatic veins and its tributaries, and is generally due to cardiac and pulmonary disease. Mitral stenosis and incompetence among

cardiac, and emphysema and cirrhosis of the lung among pulmonary diseases, together with intrathoracic tumours, are the chief causal conditions.

The liver is described by the pathological term *nutmeg*, and the symptoms are those of backward pressure in the portal circulation. Nutmeg liver may lead to cyanotic atrophy in cases of long standing.

The **Clinical Features** have been described under the results of backward pressure in heart disease of which they form a part. They include chronic gastric catarrh, sometimes with hæmatemesis, intestinal dyspepsia, enlargement and, later, cyanotic atrophy of the spleen, and ascites. As regards the liver, a slight jaundiced tinge is common, with discomfort and sometimes actual pain, while there may be enlargement of the liver, or in old standing cases actual atrophy. Sometimes pulsating liver is noted, but it is rare. There is often bile in the urine, and the stools may be somewhat clay-coloured. The spleen may be enlarged.

The **Treatment** consists in relieving the portal circulation, and in endeavouring to remove or alleviate the cause of the backward pressure whether cardiac or pulmonary. Sometimes a hæmatemesis diminishes the portal engorgement, but saline purgatives are especially useful, given in concentrated solution warm before breakfast. It is rarely desirable to bleed from the liver, although this has actually been done.

(8) Cirrhosis of the Liver

There are several different forms of this disease.

(a) Common Cirrhosis of the liver, or Alcoholic Cirrhosis, or Hobnailed Liver, is generally the result of alcohol, although other toxic agents must in certain cases play an important part.

(b) Hypertrophic Cirrhosis is believed to be due to a toxin involving the bile-ducts. It is doubtful whether this form of cirrhosis is not in reality a type of common cirrhosis.

(c) Cyanotic Atrophy or cirrhosis due to long-standing engorgement of the hepatic veins.

(d) Capsular Cirrhosis, in which the fibrous tissue spreads into the liver substance from Glisson's capsule, it is generally secondary to perihepatitis.

(c) Syphilitic Cirrhosis, in which individual cells may be surrounded by connective tissue, although larger strands are met with frequently with gummata.

(a) **Common Cirrhosis; Alcoholic Cirrhosis; Gin-Drinker's Liver; Hobnailed Liver.**—In this form of cirrhosis groups of lobules are surrounded by fibrous tissue which spreads along the lines of the medium-sized portal spaces.

Etiology.—The disease is most common in middle life, and is the result of an excess of spirituous rather than of malt liquors. There are cases, however, of cirrhosis in children, in some of whom an alcoholic history may be obtainable, but in others the etiological factor remains uncertain.

Pathological Anatomy. The liver shows a very typical appearance, and the hobnail-like surface is often palpable during life. On section the lobules are seen to be surrounded by thick bands of connective tissue, and fresh attacks are constantly made on the periphery of each little group of lobules by strands of connective tissue, gradually diminishing the liver tissue as the disease progresses. A varying amount of fatty change may be seen in the hepatic lobules which are affected, and the liver on section is often tinged with bile. During the earlier stages the liver is of almost normal size, but as the disease advances it may become markedly smaller. The connective-tissue increase interferes greatly with the portal circulation, and as a result there is backward pressure in the organs which send their blood to the portal. The spleen is enlarged and shows chronic venous congestion. The stomach and intestines may show chronic catarrhal changes, and ascites is extremely common.

It is well to remember the accessory channels by means of which portal blood can find its way into the common venous circulation.

(1) By the round and suspensory ligaments of the liver the blood may find its way into the epigastric, mammary, and other neighbouring veins, and in so doing forms the typical *caput Medusæ* seen surrounding the umbilicus in these cases.

(2) The œsophageal veins anastomose with the gastric veins at the region of the cardiac orifice, and by this means relief may be obtained for the engorged gastric veins.

(3) The portal veins communicate with the inferior mesenteric veins and form the hemorrhoidal plexus.

(4) The veins of Retzius unite the terminals of the portal veins in the intestines and mesentery with venous radicles belonging to the inferior cava, and a large quantity of blood is conveyed from the engorged portal circulation by means of these vessels.

Clinical Features.—For a long time there may be no symptoms and no discomfort experienced by the patient, but sooner or later the backward pressure in the portal system gives rise to definite and typical phenomena. These include bleeding from the stomach and more rarely from the bowels, and the development of ascites preceding in its appearance any general dropsy—in fact, œdema of the legs is only induced when the ascitic effusion increases so much as to interfere, by pressure, with the inferior cava. Hæmorrhoids are not uncommon, and the *caput Medusæ* may be seen round the umbilicus, while the spleen usually enlarges. There is nearly always a slight degree of jaundice, and very frequently a trace of albumin in the urine, while the chronic gastric catarrh renders the patient's life very miserable.

The *Physical Signs* of the ascitic effusion are referred to under ascites, but the striæ seen on the abdominal wall often far exceed both numerically and in depth those found in pregnancy. The fluid is typical dropsical fluid with no special characteristic. Bleeding from the gums, which are often spongy, is frequently complained of by the patient.

Very many patients suffer also from definite toxæmic manifestations, usually becoming restless and delirious at night, and eventually passing into a stuporose condition. Death generally closely follows the development of such symptoms.

Diagnosis.—The ascites is very suggestive, and if the patient's history includes a predilection for alcoholic excess, there is rarely much difficulty. Sometimes the hobnail-like liver is easily felt; more frequently it cannot be determined during life. The possibility of *gummata* of the liver interfering with the portal vein should not be forgotten, and often a history of syphilis aids in the diagnosis of these cases.

The **Prognosis** is unfavourable, although in certain cases remarkable benefit has accrued from repeated tapping, but where there is permanent engorgement of the portal circulation, improvement is not to be expected, and a very serious increase

in the gravity of the case occurs should a thrombus form in the portal vein.

The **Treatment** consists in tapping ascitic effusions, or sometimes in an attempt to promote collateral circulation between the omentum and the abdominal wall, while in certain cases after laparotomy the surgeon strives, by scraping or irritating the peritoneal surfaces, to induce as far as possible occlusion of the peritoneal sac.

(b) **Hypertrophic Cirrhosis, or the Cirrhosis of Hanot.**—Many authorities hold that this is merely a form of common cirrhosis, but it differs from most cases of alcoholic cirrhosis, inasmuch as the liver is greatly enlarged and is deeply bile-stained, while jaundice is at least a typical feature in the case.

Etiology.—It occurs in young persons and generally in males. Alcohol plays an unimportant part, but there is no other known etiological factor.

Pathological Anatomy.—The liver is enlarged, the surface is smooth, the colour deep green. The cirrhosis is often described as being rather monolobular than polylobular. There is a remarkable development of recently formed bile passages. The spleen is enlarged, and is often of great size.

Clinical Features.—The disease is a chronic one, beginning generally with a mild form of jaundice, but developing into a permanent jaundice, associated with pyrexia and usually delirium. There is bile in the urine, but the uric acid excretions are also well coloured. The liver is painful on pressure, and there is the typical nausea and vomiting of a stagnant jaundice. There is no ascites, but hæmorrhages are no means uncommon.

The *Physical Signs* include enlargement of the liver, which may reach to the umbilicus, and the spleen is also enlarged. The disease generally results from malignant jaundice, and there is no treatment.

c) **Cyanotic Atrophy, or cirrhosis owing to long-standing congestion of the hepatic veins.**—This form results from increased pressure often of cardiac, sometimes of pulmonary origin, and is due to an increase of connective tissue in the interlobular spaces, leading to the central or hepatic zone of the lobules. The liver is not enlarged in size, but otherwise there is no very marked

(*d*) **Capsular Cirrhosis or Perihepatitis.**—There are many diseases in which perihepatitis may occur, as, for example, syphilis or cirrhosis. As a result, fibrous tissue penetrates from the capsule of the liver into its substance, and there may be very marked interference with the portal circulation in advanced cases. There is no treatment of any special value.

(*e*) **Syphilitic Cirrhosis.**—In this disease individual cells may be surrounded by connective tissue, although larger strands, frequently with gummata, may also be met with. Gummata, which have already been referred to under syphilis generally, may cause marked scarring and fissuring of the liver, and associated with the gummata we find a fibrous-tissue increase throughout the organ, radiating from the various gummata, and leading eventually to very marked cicatrisation. Not infrequently waxy degeneration is also present.

The **Clinical Features** depend upon the interference with bile-ducts or blood-vessels, while the physical signs are often very suggestive, the fissuring of the liver being sometimes palpable to the hand.

(9) Abscess of the Liver

A localised suppuration either in the liver substance, in the blood-vessels, or in the bile passages.

(*a*) **Tropical Abscess.**—This occurs in cases of dysentery, and is the result of the local presence of the dysenteric amœba in the liver. In most cases there is a solitary abscess, although in others abscesses may be scattered throughout the organ. The abscess contains greenish-yellow or reddish pus mixed with blood, and the wall is composed of necrosed liver tissue. Where an abscess has existed for some time, a fibrous capsule may form. Other organisms, besides the amœba, may be present, and the abscess may perforate into the pleura, lung, peritoneum, or elsewhere.

There may be no clinical features until rupture occurs, but often there is pyrexia with local pain and hepatic enlargement. The fever suggests the presence of pus and may be of a hectic type. Palpation is painful, but it depends on the position and size of the abscess whether the condition can be recognised by the hand. The abscess is very frequently situated in the right lobe, but only occasionally is fluctuation detected.

A leucocytosis would be expected, but is often absent. Where the abscess perforates, grave phenomena may result; sometimes the patient expectorates the reddish or greenish viscid pus, if perforation occurs into the lung.

The **Diagnosis** is greatly aided by the presence of the amoeba in the stools and the history of dysenteric diarrhoea; the **Treatment** should consist in the opening of the abscess, often preceded by an exploratory puncture so as to localise the pus.

(b) **Suppurative Cholangitis**.—Suppuration is noted as frequent result of gall-stones, and it is termed *Suppurative cholangitis*. The abscesses are sometimes multiple, or a great deal continuous extent of the bile passages may be inflamed, the pus contained in the ducts being bile-stained. Inflammation of the bile-ducts may, however, result from other causes besides gall-stones; for example, the entrance of parasites, such as the *Ascaris lumbricoides*, or, more commonly, bacteria which are passed upwards from the duodenum.

The **Clinical Features** include hectic temperature, pain over the liver, and often severe jaundice.

The **Prognosis** is very grave, and the **Treatment** must consist in an attempt at draining the abscess.

(c) **Pylephlebitis Suppurativa**.—This is abscess formation due to infection by the portal vein; the infective agent comes from the intestine, or from some organ which sends its blood into the portal circulation. The abscesses are multiple, in fact, a large extent of the portal vein in the liver may be attended with pus.

(d) **Pyæmic Abscess**.—In this case the hepatic arteries are the channels by which the infective agent reaches the liver. The abscesses are generally multiple, and may vary in number and size.

In both pyæmic abscess and suppurative pylephlebitis, the liver is enlarged and painful, jaundice is common, and a fatal toxæmia is almost invariable, while death generally occurs, and curative interference is hopeless.

Parasitic cysts sometimes suppurate, and parasites or foreign bodies which may find their way into the bile-ducts (just as the *Ascaris lumbricoides* already mentioned), may set up a cholangitis. An injury sometimes causes abscess formation.

(10) **Fatty Infiltration and Fatty Degeneration of the Liver**

These processes are often associated. **Fatty Infiltration** is more marked in obesity; fat is also stored up in the liver cells in chronic alcoholism, and where, in the cachexia of phthisis, there is grave interference with oxidation processes. **Fatty Degeneration** is specially seen in phosphorus poisoning and in acute yellow atrophy of the liver, while it is present in localised parts of the liver in common or alcoholic cirrhosis.

In **Fatty Infiltration** the liver may attain a great size, reaching 8-12 pounds in weight, the colour is light yellow, and after section there are oil droplets on the blade of the knife. Curiously enough a liver so affected appears to discharge its functions in a wonderfully satisfactory manner. The edge of a fatty liver lacks the sharpness of waxy degeneration, and is smooth, with no nodules or cicatrices suggestive of cancer or syphilis. The skin is peculiarly greasy.

In **Fatty Degeneration** the organ is small.

In many cases, where limited fatty infiltration and degeneration occur together, there are no symptoms.

The **Prognosis** depends on the nature of the case. Fatty infiltration, if the result of obesity, is within the sphere of treatment.

The **Treatment** for obesity is given elsewhere (p. 163), but it is, in brief, a dry diet with little fatty or fat-forming food, and a sufficiency of exercise suited to the patient's age and strength.

(11) **Waxy or Amyloid Degeneration**

The causes of waxy degeneration in other organs determine its appearance in the liver. These are syphilis, suppuration of a prolonged nature, and bone disease. The disease is seen first in the middle zone of the lobules, where it attacks the capillary walls, but it spreads to the interlobular vessels. The liver is of great size, 8-14 pounds, and has a smooth sharp edge (unless gummata are present, when cicatrices may cause indentations).

There are no **Clinical Features** referable to the disease in the liver, although the co-existence of enlargement of the spleen

and evidence of waxy involvement of the kidneys and intestine help materially in the diagnosis.

The **Prognosis** is unfavourable, and nothing can be done by way of treatment, although the presence of waxy degeneration should make the physician cautious in ordering cod-liver oil and fatty articles of diet.

(12) Hydatid Cyst of the Liver

The liver is the commonest site for a hydatid cyst. The cyst or cysts may be in any part of the organ, but more commonly in the right lobe, and may grow to a large size. If superficial, the hydatid thrill may be obtained on palpation. Sometimes the cyst pushes up the diaphragm and greatly increases the liver dulness upwards, in other cases the rounded cyst wall is palpable below the edge of the liver. Occasionally cysts suppurate and give rise to the clinical features of a hepatic abscess. Cysts may perforate into pleura or peritoneum, but are generally diagnosed and treated by tapping or itching the cyst to the wound and draining.

(13) Tumours of the Liver

These may be simple, such as *angiomata*, which are fairly common, and occur as small vascular tumours about the size of a walnut. Malignant tumours are the most frequent, and generally cancerous and secondary to a tumour elsewhere.

The **Etiology** of malignant tumours is a very difficult problem. The liver is the third organ in the body most commonly the site of such tumours, and in it they are, as already stated, generally secondary. Probably gall-stones play a very important part in the etiology of hepatic cancer, the disease commencing in the gall-bladder or bile-ducts. Injury may possibly be associated in some way with primary hepatic tumours, but it is easy to understand how a primary tumour of the stomach will readily induce secondary growths in the liver.

The Varieties of Malignant Tumour

1. **Cancer** may be **Primary**, but is far more frequently **secondary**.

Primary Cancer is described as occurring in three forms.

Either (*a*) a nodular cancer scattered throughout the liver, one of these nodules, which may be larger, being the primary tumour. This form is almost indistinguishable from the common form of secondary cancer. Or (*b*) a massive cancer in which there is a huge, but sharply defined, mass of primary cancer which involves often a large extent of the liver. Or (*c*) a rare form with much cirrhosis, and which may be found in an organ which is not enlarged in size.

Secondary Cancer.—The primary sites are the stomach, in fully a quarter of all cases, the gall-bladder, the rectum, and the cervix uteri in women; women suffer rather more frequently than men (4 to 3).

The liver is often of a huge size, even reaching 20 pounds. The masses vary greatly, from the size of a small pea to a cocoa-nut, and may be very numerous. The older ones undergo fatty degeneration centrally, and as a result umbilication is often well marked in peripheral nodules; this characteristic feature may be felt on palpation. There is often perihepatitis, and not infrequently the diaphragm becomes infiltrated, or the abdominal peritoneum infected, nodules appearing first in the neighbourhood of the umbilicus.

The tumours may press upon bile-ducts and lead to local dilatations, or may interfere with vessels.

2. **Sarcoma** may be **primary** or **secondary**.

Primary Sarcoma is very rare.

Secondary Sarcoma is more frequent. One very marked variety is the so-called melanotic sarcoma, which should rather be termed a cancer. The primary tumour is either in the choroid of the eye or in the skin. Melanotic sarcoma may cause very great enlargement of the liver, and the nodules of new growth have varying shades of pigmentation. In other cases of secondary sarcoma the liver may be infiltrated with many nodules.

The **Clinical Features** vary with the number, size, and position of the tumour nodules. Generally speaking the liver is enlarged, often greatly, and there are evidences of cachexia, with gradual, sometimes rapid, emaciation, loss of appetite, often nausea, vomiting and jaundice. The degree of jaundice depends on whether nodules in the transverse fissure of the

liver obstruct the bile-ducts, and such jaundice, once established, is apt to be permanent. Pain varies; sometimes it is better described by the term uneasiness, in other cases there is much actual sharp pain. Friction where perihepatitis is present not infrequently causes pain. Occasionally the temperature rises to 100° or 101° F. Very often the feet swell, partly owing to the anæmia, partly to interference with the inferior vena cava.

The *Physical Signs* are usually definite. On *Inspection* the enlarged liver may be visible moving downwards with the diaphragm on inspiration, and even projections, due to nodules of new growth, may be seen, and sometimes dilated veins indicate interference with portal or systemic veins. *Palpation* reveals the enlarged liver and frequently the presence of umbilicated nodules. *Percussion* corroborates the enlargement, and on *Auscultation*, friction, due to perihepatitis, may occasionally be heard.

The **Diagnosis** largely depends on the possibility of palpating the umbilicated nodules, the existence of a primary tumour in stomach or elsewhere, and the cachexia and permanent jaundice (if present). *Gummata* have occasionally led to a mistaken diagnosis, but there is often a history to assist, and antisyphilitic treatment yields beneficial results not obtainable in cases of tumour. Time, too, usually clears up the diagnosis in a doubtful case.

The **Prognosis** of tumour is always grave, and the duration of life varies with the rapidity of growth of the neoplasm, but it is rarely longer than a year, and often much shorter.

The **Treatment** is purely palliative. It is necessary, where pain is present, to give morphia, and to apply fomentations or poultices locally.

(14) Cancer of the Gall-Bladder and Bile-Ducts

Cancer of the Gall-Bladder may be primary, and is probably due to the irritation of gall-stones (fully 90 per cent. cases). There may be a history of preceding biliary colic, and as the tumour is apt to spread secondarily to the liver, the clinical features are indistinguishable from cancer of that

Cancer of the Bile-Ducts is rarely primary, but may in such cases be due to gall-stones. It causes marked interference with the bile-ducts, with consequent dilatation and permanent jaundice.

It is possible that surgical interference, either draining away bile from the gall-bladder into the intestine, or making an external biliary fistula, may in certain cases afford much relief.

(15) Diseases of the Portal Vein

1. **Passive Hyperæmia** has already been described.

2. **Thrombosis of the Portal Vein, or Pylephlebitis Adhesiva**, occurs specially in cancer when it involves the veins, and in alcoholic cirrhosis of the liver. The clot may be extensive or limited, and it may only affect a part of the vein at any one site. The affected part of the vein may become completely occluded and form a fibrous thread—hence the name Pylephlebitis adhesiva. In order to carry on the portal circulation collateral anastomosis may provide channels, but if these fail or are insufficient, ascites and hæmatemesis are inevitable, in really extensive cases.

Emboli in the portal circulation do not cause infarction owing to the free anastomosis with the hepatic artery.

3. **Suppurative Pylephlebitis** is described under Abscesses of the Liver.

(16) Affections of the Hepatic Vein

1. **Passive Hyperæmia** has already been described with the consequent portal engorgement.

• 2. **Emboli** may, in rare cases, enter the hepatic vein; equally uncommon is it to find—

3. **Phlebitis** or other interference with the hepatic vein, unless from accidental involvement by tumour.

XVII. DISEASES OF THE PANCREAS

(1) Acute Pancreatitis

THE most common variety of this is **Acute Hæmorrhagic Pancreatitis**. It is rare, although cases are met with from

time to time in the post-mortem department of our large hospitals.

Etiology.—It is more frequent in men during adult life, and is said to depend, in certain cases, on alcoholism.

Pathological Anatomy.—The pancreas shows many hæmorrhages on section, and, at operation or post-mortem examination, little areas of fat necrosis may be often seen all over the abdomen, in the omentum and mesentery. Fat necrosis is due to the action of pancreatic secretion on fat, and its presence implies an escape of the secretion into the general peritoneal cavity.

The **Clinical Features** are fairly typical. The patient is suddenly seized with violent colicky pain in the upper part of the abdomen, and this is followed by nausea and vomiting and the early appearance of symptoms of collapse, death often occurring in two to four days with the abdomen distended and rigid. There may be definitely greater resistance in the region of the pancreas, and there is, certainly, greatly increased sensitiveness to pain upon pressure.

The **Diagnosis** lies between *intestinal obstruction* and acute pancreatitis, and a careful note should be made of any point in the history suggesting obstruction. In acute pancreatitis the condition is sudden, and comes on, often, in a previously healthy individual, the localised area of pain and resistance suggesting the pancreas. At the operation, in a case of obstruction, we find below the stricture coils of intestine empty of gas, while in acute pancreatitis there are no depleted coils, and we note also the patches of fat necrosis.

The **Prognosis** is bad, but a few cases have recovered.

The **Treatment** is purely expectant. Operation is too often futile; pain can be soothed, and either heat or cold applied locally, while the patient's strength is supported with stimulants.

(2) Suppurative Pancreatitis

This rare disease is sometimes the result of trauma, but more often no cause can be assigned for it. There may be one, or several, suppurative foci. The clinical features vary; there may be local pain and vomiting, and sometimes definite resistance can be made out, on palpation, over the site of the pancreas. There may be fat in the stools, and sometimes sugar

in the urine, and the inflammation may spread and set up jaundice. The abscess may open into a neighbouring viscus, and occasionally the portal vein becomes infected, causing thrombosis, and possibly pylephlebitis suppurativa (abscess formation in the liver in the line of the portal circulation).

The disease often tends to run a chronic course, and the only possible treatment is surgical, an attempt being made to drain the abscess.

(3) **Gangrenous Pancreatitis**

This may result from either of the preceding diseases or from injury. It is nearly always fatal. The clinical features are very acute and resemble hæmorrhagic pancreatitis. In one or two reported cases the necrotic pancreas has been passed by the bowel into which it had perforated.

(4) **Cirrhosis and Atrophy of the Pancreas**

There may be either (A) a thickening of the interstitial septa of the gland, or (B) the fibrous tissue may penetrate and involve the glandular structures themselves, namely, the islands of Langerhans. Both these conditions lead to hardening of the organ, and they may be combined. In diabetes there is often not merely an atrophy of the pancreas, which is a common condition in that disease, but the islands of Langerhans are specially affected by sclerosis. A chronic interstitial fibrosis of the pancreas has been known eventually to involve the ductus choledochus and thus cause jaundice.

(5) **Pancreatic Concretions or Calculi**

These are rare and are very difficult to diagnose, although, when they are passed by the patient, or cause the formation of a cyst, their presence may be confirmed. Men suffer twice as frequently as women. These calculi occur in the duct of Wirsung, the branches of which are dilated. They are of a whitish colour, may be coral-like in structure, and consist of calcium carbonate or phosphate. Occasionally they cause suppuration and sometimes colic, but not as a rule. There may be fat in the stools owing to the blockage of the main duct, and indicating loss of the emulsifying ferment, and there

may also be glycosuria, due to interference with the internal secretion of the organ.

There are often no clinical features except in severe cases, and the treatment, where the calculi are diagnosed and are giving trouble, is surgical.

(6) Pancreatic Cysts

Besides hydatid cysts, and cysts in the pancreas associated with cystic disease in the kidneys, liver, and brain, all of which are rare, there are cysts which are very difficult to explain. Plugging of the duct of Wirsung does not necessarily, or even commonly, cause cystic distension of the duct behind the block, and although calculus and tumour may explain a certain number of retention cysts, they do not explain the larger and more important ones. There may be no block at all. Some of these cysts seem explicable as the result of injury, but even this is disputed; perhaps some of them are cystic adenomata formed in connection with the gland structures.

These cysts are generally of large size; the fluid is turbid, alkaline, and albuminous, and may possess the properties of a dilute pancreatic secretion. The cyst may contain 400 oz. of fluid, and so form an immense abdominal tumour. It may grow into the lesser sac of the omentum, and, pushing the stomach upwards, may appear between the stomach and transverse colon, or it may push the stomach downwards; it much depends upon the part of the pancreas with which it is connected and from which it grows. The cyst will in such cases be palpable, and is found to be fixed, and not movable with the diaphragm. The chief symptoms are due to the cyst pressing on the stomach and other neighbouring organs, and include dyspepsia and sometimes vomiting. There may, but rarely, be fatty diarrhoea; glycosuria is more common, and generally there is rapid emaciation. Hæmorrhage sometimes pours into the cyst, and may lead to alarming collapse.

The **Treatment** consists either in tapping the cyst, an operation which may need to be repeated, or else in freely opening below the 12th rib and draining. The presence of persistent glycosuria is a grave feature.

(7) **Tumours of the Pancreas; Malignant Tumours**

These may be primary or secondary.

I. **Primary Tumours**.—(1) Carcinoma of the scirrhus variety is described as being not infrequent and as involving the head of the pancreas. In our experience it is extremely rare, and, with two exceptions, the cases at first believed to be primary tumours of the head of the pancreas were found not to involve the gland primarily. (2) Sarcoma is very rare.

II. **Secondary Tumours** are much more common, both carcinomatous and sarcomatous. Often the so-called primary carcinomas of the head of the pancreas are really secondary, and certainly involve by preference the head of the organ.

The **Clinical Features** are generally distinctive, and include fatty diarrhoea, sometimes glycosuria, usually epigastric pain, jaundice due to the involvement of the orifice of the ductus choledochus, a palpable tumour which is painful on pressure, and rapid emaciation. Sometimes the pylorus is interfered with and gastrectasis develops.

The **Diagnosis** is in many cases fairly easy.

The **Prognosis** is bad.

The **Treatment** is simply that of symptoms, pain often demanding the use of opium.

Gummata are sometimes met with in the pancreas, and occasionally tubercular tumours.

XVIII. DISEASES OF THE PERITONEUM

(1) **Acute Inflammation of the Peritoneum**

Etiology.—Acute peritonitis may be of **Primary** or **Secondary** origin.

Primary cases may be dismissed in a couple of words. They are distinctly rare: although called primary, they are the result of an organism or possibly a toxin, and occur in affections which are, more or less certainly, of infective or toxic nature, such as influenza, Bright's disease, sometimes acute rheumatism, and occasionally gout. It is probable that,

in every case, an organism is present. It is extremely unlikely that cold can alone be responsible for such a condition.

Secondary peritonitis is by far the more important. It may be of local origin, and may be associated with a lesion of the stomach or some part of the intestine, abscess in the liver, glandular suppurations, pyosalpingitis, or other pelvic suppuration. Among the most common varieties of local peritonitis in connection with the intestine is that due to appendicitis. Many cases are generalised from the outset, where the inflammation is not limited by adhesions, but perforation through the posterior wall of the stomach may involve only the lesser sac of the omentum if the foramen of Winslow is closed, whereas perforation through the anterior wall of the stomach will almost certainly set up a general peritonitis. Malignant tumours of stomach or bowel are sometimes responsible for the involvement of the peritoneum. Perforation of a suppurating gall-bladder and the rupture of an extra-uterine gestation constitute two possible, though not very common, causes of peritonitis.

Acute peritonitis may be associated with the distribution of acute miliary tubercles over the peritoneum, and in pneumonia, septicæmia, and pyæmia the peritoneum, as well as other serous membranes, may become infected.

Of 102 cases referred to by Professor Osler, 56 were due to extension from some abdominal focus, 34 followed surgical procedures, and 12 were described as primary, and in all of these 12 an infective organism was present.

Pathological Anatomy.—The peritoneum becomes intensely congested, and both adhesive and serous or sero-purulent lymph is rapidly poured out. Often matting together of loops of the bowel occurs. The bowel becomes distended, and in a severe case the muscle in the wall of the gut is paralysed, causing complete obstruction, because of the interference with ordinary peristaltic contractions as well as from the presence of adhesions. The fluid may amount to from 20 to 500 or more ounces; it may be sero-fibrinous or purulent, or in certain cases hæmorrhagic, and sometimes it has a fecal colour. Occasionally the bowel and affected peritoneum become gangrenous.

The following organisms may be present: the *Streptococcus pyogenes* and the *Bacillus coli communis*, which are extremely

physical examination. Where there is air or gas in the peritoneal sac, the liver dulness may completely disappear.

(B) **Localised Peritonitis.**—In this case the pain is generally limited to the side of the affection, and while it may occur in many different positions, it is perhaps more frequent either in the region of the appendix, in the pelvis, or in the lesser sac of the omentum, and the most careful examination is often necessary to enable a diagnosis of the latter site of peritonitis to be made. A subphrenic abscess is a not infrequent result of peritonitis in the lesser omental sac, and the fact that the liver is displaced downwards is suggestive. In localised cases of peritonitis, the symptoms, as a rule, are less severe, although there may be the same vomiting, intense pain, high temperature, and eventual death. One great risk is that the abscess may burst into the general peritoneal sac, while in certain cases it opens into the bowel, or may even make its way externally.

Forms of Localised Peritonitis.—(1) *Appendicular Peritonitis.*—In this case a localised peritonitis, or possibly a peritonitic abscess, may be associated with the inflamed appendix. The clinical features are those of appendicitis, and the position of the abscess is generally just behind the termination of the ileum.

(2) *Pelvic Peritonitis.*—Beyond stating that this form of peritonitis is much more common in women, and is generally due to salpingitis, it is hardly necessary to describe it here.

(3) *Subphrenic Peritonitis.*—Peritonitis may be localised to a part of the greater peritoneal cavity where it is related to the liver, or it may be confined to the lesser omental sac, and in cases of cancer or ulcer of the stomach or pancreatic inflammations, abscesses confined to this sac are by no means uncommon. Sometimes the subphrenic abscess contains gas or air.

The **Clinical Features** of subphrenic abscess vary considerably: they include marked constitutional symptoms, and suggest sometimes merely localised pain, but sometimes the presence of a considerable effusion of pus. The breathing may be seriously interfered with, and physical examination demonstrates the displacement of the liver downwards by the effusion. The symptoms depend, in part, on the cause

of the condition. Unfortunately few cases of subphrenic abscess terminate favourably.


The **Diagnosis** of peritonitis.—Where the peritonitis is general, the clinical features are extremely characteristic, and the difficulty consists rather in the recognition of cases of acute localised peritonitis which may in time become generalised. In females, the possibility of a salpingitis should not be forgotten, and perforating appendicitis and gastric ulcer should be kept in mind. In typhoid fever ulcers sometimes perforate, but there is, as a rule, little difficulty in the diagnosis.

In *fecal impaction*, or other form of *intestinal obstruction*, and in cases of *enteritis* the pain may suggest peritonitis, but in these cases it resembles ordinary colic, and in enteritis, at all events, diarrhoea is common. In *hysterical* cases the pain of peritonitis may be simulated, and the typical picture of a peritonitis, if previously familiar to the patient, may be portrayed with great exactness, and it is only from the knowledge of the patient's previous history and by careful observation of the case that a true diagnosis can be made.

The **Prognosis** is always grave in every case of peritonitis, generalised or localised, and in cases where a gastric or intestinal ulcer has perforated, prompt surgical treatment can alone yield any hope of cure.

Treatment of Acute Peritonitis.—In every case an attempt should be made to diagnose the cause of the condition, so that, where necessary, the patient may be operated on with the least possible delay.

The medical treatment of acute peritonitis consists in rest in bed with a cage over the abdomen, so as to remove the pressure of the bedclothes. Decide next whether to apply ice, or to use warm applications in the form of fomentations with lead and opium, or poultices. In many cases ice affords the greatest relief, while it prevents, to a certain extent, the awful vomiting which distresses the patient. Morphia should also be administered hypodermically to relieve pain. Where abdominal distension is very extreme, a long rectal tube may be passed into the bowel so as to draw off gas, or the abdominal wall may be punctured, and the gas taken directly from a distended coil. Sometimes the use of turpentine or assafoetida enemata gives relief in these cases. Lastly, for the



sickness and nausea the stomach may be occasionally washed out.

The different forms of localised peritonitis demand treatment suitable for each condition, and beyond stating again that the ice-bag or the poultice may be applied locally, and that morphia hypodermically relieves pain, nothing need be added here.

(2) Chronic Peritonitis

A chronic inflammation of the peritoneum which may be general or localised.

(a) **Cancerous Peritonitis.**—This is usually *secondary* to cancer of the stomach or other organ, but *primary* peritoneal tumours, most of which are endotheliomata, are sometimes seen. In the majority of secondary cases, in addition to the primary lesion, there are numerous cancerous nodules, varying greatly in size, dotted over the peritoneum. There are many adhesions, while the mesenteric glands are enlarged, and the omentum is often puckered up, forming a large mass of new growth stretching across the abdomen. The amount and kind of the fluid present vary in different cases. It is often hæmorrhagic, and sometimes owing to secondary infection it becomes purulent.

In colloid cancer the disease infiltrates the peritoneum to remarkable extent, and large quantities of jelly-looking material are present in the peritoneal sac.

The **Clinical Features** vary with the position of the primary lesion and the interference due to the peritoneal involvement. The presence of fluid can readily be made out, and the physical signs are referred to under ascites. Intestinal obstruction is a frequent sequel, and the amount of sickly pain is often very great.

In the **Diagnosis** the cellular elements in the fluid should be examined; they may suggest the cancerous origin. The presence of an omental tumour is very striking, and cancerous nodules near the umbilicus frequently follow carcinoma of the stomach.

The **Prognosis** is grave in the extreme.

(b) **Tuberculous Peritonitis.**—There may be either an *acute miliary tuberculosis*, often with the presence of hæmorrhagic effusion, or (2) *tubercular masses of a large size*

causing great matting of the bowel, although in some cases what may be described as (3) a *chronic miliary tuberculosis*, with comparatively few large tumour masses, may also be met with.

The tubercular involvement of the peritoneum is often secondary to, or associated with, tubercular ulceration of the intestine, and in many cases the mesenteric glands are also extensively involved. Not infrequently, persons so affected have obvious pulmonary disease, or the glands in the neck are tubercular. While tubercular peritonitis must be described alone, it should be remembered that it generally occurs coincidently with tubercular enteritis and *tabes mesenterica*. Tubercular peritonitis is most common in children, and is certainly rare after middle life.

The **Clinical Features** vary greatly, and depend much on the size of the tubercular tumours, the amount of matting of the intestine, and the presence or absence of ascites. In certain cases there is marked pyrexia of hectic type, with great abdominal tenderness, while in others there is little temperature and a comparatively small amount of pain. Intestinal dyspepsia is common, and the abdomen is often swollen, more from the presence of gas in the intestinal coils than from the fluid, which may also, however, be in considerable amount. In some cases the omentum forms a definite tumour infiltrated with tubercular masses, and its position, stretching across the abdomen, is very characteristic. Effusions are often sacculated, and definite tumour-like masses may be felt in different regions of the abdomen, while in many cases the enlarged mesenteric glands may be made out on careful palpation. These mesenteric glands are peculiarly common in the younger patients, and are tender to the touch.

There may be very acute cases of tubercular peritonitis, and also extremely chronic types which closely simulate other affections.

In **Diagnosis** it is desirable to investigate the family history, and to examine with the greatest care for evidences of tubercular involvement of the chest. It should not be forgotten that in females a primary tuberculosis may be met with in the Fallopian tubes, and that a tubercular salpingitis can sometimes be definitely recognised by vaginal examination.

(c) **Proliferative Peritonitis**.—In this affection the peritoneum becomes greatly thickened, in fact, undergoes

proliferative changes, and, as a result, the mesentery is shortened and the omentum pulled upwards, forming a more or less definite tumour. The thickening also affects the peritoneal covering of the liver and other organs, and there is frequently perihepatitis or perisplenitis. This thickening may be due to frequent tapping of ascitic effusions, but there are many cases in which the etiological factor is by no means clear. It occurs along with cirrhosis of the liver, and occasionally in alcoholic subjects.

The **Clinical Features** are sometimes obscure. There is generally considerable ascitic effusion, and not infrequently the localised peritoneal thickening interferes with the intestine, causing obstructive symptoms.

(*d*) **Local Chronic Peritonitis**, not included in the above three groups, is very common in connection with the liver and spleen, and also, though less commonly, with other abdominal organs. It may occur in cirrhosis of the liver or syphilitic disease of that organ, and it is not uncommon in carcinoma hepatis. Perisplenitis is frequently associated with leucocythæmia, and is also a common result of splenic infarction.

The **Clinical Features** include pain, often with definite friction, palpable and audible, while bands of adhesions may constrict a loop of bowel and thus cause acute obstruction.

Treatment of the Foregoing Conditions.—In *Malignant* cases little can be done except to relieve the patient by occasional tapping and by local and internal sedatives. *Tubercular Peritonitis* yields satisfactory results, often simply from abdominal section, the operative interference apparently blighting the disease. Medicinal treatment includes the use of mercurial or potassium iodide ointments rubbed into the skin of the abdomen and sometimes applied on a binder. Careful feeding is advantageous, and the better the condition in which the patient's general health can be placed, the more likely will improvement result.

Proliferative Peritonitis is incurable, although the removal of fluid when necessary adds greatly to the patient's comfort. *Local Chronic Peritonitis* should be treated either by the application of the ice-bag or by counter-irritation.

The method of performing the operation of tapping the peritoneum is described under ascites.

(3) **Ascites**

The effusion of more or less serous fluid into the peritoneal sac.

It is due to a variety of conditions, and is in reality rather a feature of several diseases than a disease in itself.

Etiology.—**A.** The fluid may be a *transudation* or dropsical lymph due to (1) backward pressure from heart disease; lung disease, as in very marked emphysema; affections of the portal circulation, especially in the liver, as in common cirrhosis of that organ; and tumours pressing on the inferior vena cava. (2) It may occur in the dropsy of Bright's disease, profound anæmia, etc.; and (3) it may result from obstruction of the lymphatic channels and thoracic duct.

B. The fluid may be an *exudation* of more inflammatory lymph, such as occurs in tubercular peritonitis and other acute and chronic forms of peritonitis, and in connection with cancerous growths of the peritoneum.

The fluid differs much in appearance and character in the two types of cases. A transudation or dropsical effusion has a low specific gravity, rarely exceeding 1012, and often about 1008, while in an inflammatory effusion it is rarely under 1018. Dropsical effusions or transudates are yellow or greenish, do not coagulate spontaneously, and contain few cells, while inflammatory effusions or exudates coagulate spontaneously, and contain a much larger number of cellular constituents. The fluid may become chylous from rupture of lacteals, as in filariasis, and it is not infrequently chylous or milky in tubercular and malignant peritonitis, owing, it is thought, to fatty degeneration of the cells lining the peritoneal sac, thus causing the milky appearance. The amount of the fluid varies greatly, from 10 or 20 oz. up to 400, 600 oz., or more. There is probably always some fluid in the peritoneal sac, but transudation and absorption balance each other; in disease this balance is upset. There is often blood in the fluid in tubercular and malignant cases, but this is not always to be depended upon; sometimes in malignant disease the cells in the fluid may be diagnostic. The peritoneum loses its smooth, glistening appearance, and in chronic cases becomes thickened and more opaque.

The **Clinical Features** depend on the amount of the fluid,



and in what is termed globular ascites, where the dropsy is of portal origin, the appearance of the abdomen with, in the first instance, no œdema of the legs, is very typical and suggestive. The fluid tends to gravitate, and the intestinal coils generally float on the top. In a marked case on *Inspection* the skin may show recent striæ or cracks, the umbilicus may be protruded like a hernia, and there may be enlarged veins indicative of the position of the pressure, either laterally, carrying blood from below up to the superior cava by the mammary veins (pressure on inferior vena cava), or by the distended veins round the umbilicus, the so-called *caput Medusæ*, by which portal blood reaches the superficial abdominal veins by veins in the round ligament of the liver. In moderate effusions there is marked flattening in the flanks.

On *Palpation* a very definite thrill or fluctuation can be made out, if the fluid is not loculated. One hand is placed over one flank, and with the fingers of the other hand the opposite flank is flipped or tapped so as to produce a wave in the fluid. As the air-containing bowel sometimes transmits a pseudo-fluctuation, it is a great help to get the nurse to place a hand vertically, in the middle line of the abdomen, by which means this pseudo-fluctuation is prevented. Often by palpation a tumour, or enlarged liver, or other condition which bears an important relationship to the ascites, is revealed. On *Percussion* the fluid is found to follow the law of gravity, unless there are adhesions, so that with the patient in the dorsal position, the flanks are dull, and the intestines floating on the top of the fluid give a tympanitic note in the middle line, and it is only necessary, after percussing the flank and obtaining a dull note, to roll the patient on to his side and so get a clear tympanitic note where previously there was dullness. If the fluid is small amount, place the patient in the knee-elbow position in bed, and percuss the most dependent part of the abdomen.

Only in very rare cases does the fluid cover over the intestinal coils entirely, and especially when, as the result of proliferative peritonitis, the intestinal coils are bound back to the posterior abdominal wall.

Diagnosis.—The fact that the fluid alters its position promptly distinguishes it from fluid confined in a cyst, such as in ovarian tumour or hydatid cyst. The nature of the jelly-like,

brownish fluid in an ovarian cyst is very distinctive, and its specific gravity is generally high,—while a hydatid cyst contains clear watery fluid rich in sodium chloride, with little or no albumin, and often hooklets.

A mistake should never be made in connection with a *distended bladder*. The dribbling of urine is suggestive, and the passage of a catheter saves all further doubt.

The **Prognosis** must depend largely on the cause of the condition. Many inflammatory and some dropsical effusions are of very grave significance.

The **Treatment** varies with the nature of each case. It is often desirable, as well as necessary, to tap the effusion, but only a small amount may need to be removed so as to aid nature. Southey's tubes are generally used; sometimes, and certainly for effusions which do not flow freely, Potain's aspirator is preferable. Draw off the fluid slowly if an aspirator is used, and keep up abdominal pressure by means of a bandage, so as both to obviate shock and prevent the abdominal blood spaces distending, and the patient being bled into them with consequent fainting. Try diuretics, purgatives, and sometimes diaphoretics, and thus aid nature so far as possible, even if tapping may be also necessary. A dry diet is good practice in many cases, and it has been found that the elimination of sodium chloride is sometimes helpful.

In cases of common cirrhosis of the liver, patients have had enormous amounts of fluid drawn off, and occasionally with good results, the ascites gradually diminishing thereafter.

Many times the patient's own weight may thus be removed in an illness of months or years. An abdomen may fill up at the rate of at least 10-15 oz. a day, with a consequent heavy drain of albumin, and hence many physicians prefer to delay tapping save when it is absolutely necessary. Never tap until the bladder has been emptied, and introduce the trochar and canula in the middle line and well below the umbilicus.

DISEASES OF THE HÆMPOIETIC SYSTEM

I. GENERAL ANÆMIA

ANÆMIA may be primary or secondary.

1. Primary Anæmia

A, Chlorosis ; B, Pernicious Anæmia. — These are described separately, and it is possible that there are cases which should be termed primary anæmias, and which yet do not fall under either of these two types.

A. Chlorosis ; “ Green Sickness ”

A form of anæmia in which, with a considerable diminution in the number of red cells, there is a proportionately greater decrease in the amount of hæmoglobin.

Etiology.—This is the typical anæmia of girls at puberty, and is probably dependent on unsatisfactory conditions, as regards bedroom, work, and diet, which influence the general health at a time when the strain of development and the advent of menstruation demand the best hygienic surroundings and the greatest care of the general health. Many delicate girls are, however, exposed to insanitary surroundings at puberty, and only a small proportion become chlorotic. Constipation has, therefore, been considered a possible cause, with a resultant auto-intoxication, but many lifelong sufferers from constipation escape chlorosis entirely.

Sexual derangements may be associated, both as cause and effect. On the other hand there are cases which occur years after puberty, and there are also cases associated with imperfect development of uterus and ovaries.

A hypoplasia or maldevelopment of the heart, and especially the aorta, has been described in a few exceptional cases of the disease, but this is too rare to be accredited with an important bearing on chlorosis generally.

In conclusion, the disease is, in all probability, a defective blood formation, due largely to insanitary conditions, with unsatisfactory diet, and want of exercise and fresh air, at a time of life when development is most rapid.

Pathological Anatomy.—(1) The red cells fall to 2,000,000 or 3,000,000 per cubic millimetre of blood; and (2) the hæmoglobin index falls to a proportionately much

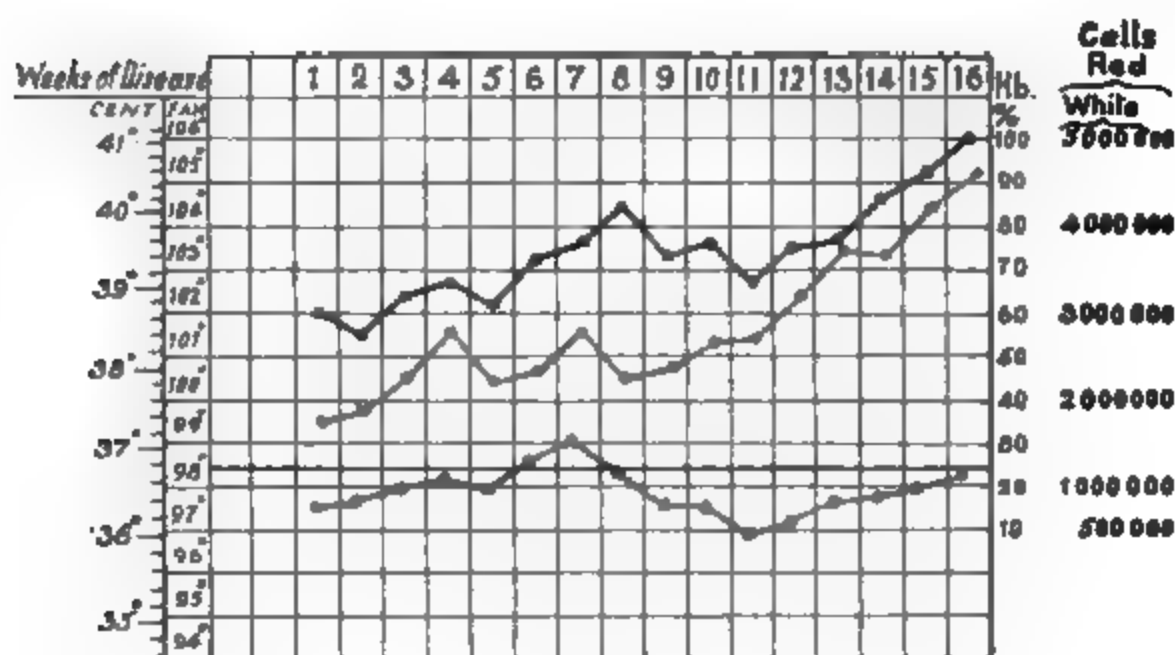


DIAGRAM 22.—Blood Chart. Case of Chlorosis. Recovery. Black line indicates number of red blood corpuscles. Red line indicates hæmoglobin index. Blue line indicates number of leucocytes.

lower figure, perhaps 20-30 per cent or thereby. There may be a degree of poikilocytosis in profound cases, but no constant alteration in the leucocytes. A few nucleated red cells may be present, indicating nature's attempt at remedying the defects.

Fatty degeneration of the heart muscle is not uncommon in protracted cases, and a similar change may occur in the liver. A remarkable feature is the plumpness of patients who are the subjects of chlorosis. A limited oedema of dependent parts is often present.

Clinical Features.—The typical changes in the blood have been described under the pathological anatomy. Patients suffering from chlorosis are usually plump, but have a peculiar

waxy-looking complexion, and often, as the name implies, may show a somewhat greenish tinge. The pallor is specially obvious in the palpebral conjunctivæ, and in the gums and lips, while occasionally a somewhat deceptive coloration of the cheeks is present. The sclerotics have a markedly bluish colour. The patient complains of breathlessness, increased on the slightest exertion, and there is langour and inability both for physical and mental work. The face is sometimes puffy, and there may be slight œdema of the ankles.

Circulatory System.—The most important point is to examine for evidence of cardiac dilatation, and a systolic murmur may be heard perhaps most distinctly and commonly at the base of the heart, often also in the mitral area.

The hæmic murmur may be explained by the theory that there is an atonic condition of the heart wall and of the fibres which surround the mitral orifice, thus leading to inadequacy so long as the anæmia persists. Its production at the pulmonary orifice may be due to unequal tension of cusps or of the arterial wall.

A *bruit de diable*, or humming-top murmur, is audible over the jugular vein at the root of the neck; in some cases it is a continuous hum, in others there is marked systolic accentuation. The *bruit de diable* is due, in part, to pressure of the cervical fascia on the bulb of the jugular vein, and it is louder when the head is turned away from the side which is being examined, because under these circumstances this pressure is increased.

The pulse is full, of low tension, and shows a tendency, on occlusion of the vessel with the finger, to the appearance of distal pulsation, owing to dilatation of vessels in connection with the palmar arch. There is a predisposition to thrombosis of veins, especially in the legs, and to a less extent the cerebral sinuses. There may sometimes be a hæmic bruit heard over the eyeball or over the lateral sinus.

Alimentary System.—There is generally loss of appetite with gastric pain, sometimes vomiting, but almost invariably persistent constipation. There seems to be a relationship between hyperacidity, so often present here, and the development of simple gastric ulcer. In certain cases the appetite is capricious, the patient longing for unusual articles of food.

In the other systems we find typical features, and especially amenorrhœa or dysmenorrhœa, and very frequently

headache, giddiness on rapidly assuming the erect posture, mental irritability, and even hysteria.

Diagnosis.—The examination of the blood is distinctive, although it may be difficult in certain cases to exclude the possibility of the condition being a *secondary anæmia*, and tubercular disease must be thought of in this connection.

Prognosis.—In the majority of cases, recovery can be definitely promised within a period of one or two months, but it should be remembered that what may be termed predisposing causes may bring about more than one relapse.

Treatment.—Try to eliminate any definite causal factor, and give iron, especially in the form of the protosalts; perhaps some preparation of ferrous carbonate is the best. Whatever salt of iron is administered, do not neglect the use of purgatives. The diet should be simple and nutritious, and the patient should have plenty of fresh air and sunshine, while in cases of cardiac dilatation, absolute rest in bed is an important part of the treatment. On no consideration allow the patient to return to work until the cardiac dilatation has disappeared and the hæmoglobin has returned to its normal proportions.

B. Pernicious Anæmia

A form of primary anæmia in which there is marked diminution in the number of red blood corpuscles and also of hæmoglobin, the hæmoglobin index for each red blood corpuscle being, however, greater than in any other form of anæmia. There are all the clinical features of profound anæmia with, in most cases, a rapid and fatal termination.

Etiology.—The following diseases appear to bear a certain causal relationship to pernicious anæmia—the presence of the tape-worm *Bothriocephalus latus*, *Anchylostoma duodenale* or anæmia worm, and gastric dilatation and tumour. An unusually profound type of any other anæmia may eventually take on a pernicious tendency.

Hunter considers that the disease is due to the entrance of organisms by the mouth in connection with carious teeth, while other authorities consider that intestinal sepsis has a direct bearing on the condition. After a prolonged hæmorrhage, severe attack of fever, or excessive physical strain,

pernicious anæmia has sometimes originated, although the causal relationship between the preceding conditions and the anæmia has not been proved.¹ The disease must be one affecting the blood-forming glands. It is rather more common in men, and about middle life.

Pathological Anatomy.—The *blood* shows a marked diminution of red cells, the number often falling to 1,000,000 or 500,000, while in exceptional cases the figure may fall below 200,000 per cubic millimetre of blood. There is marked poikilocytosis, by which is meant a distortion in shape

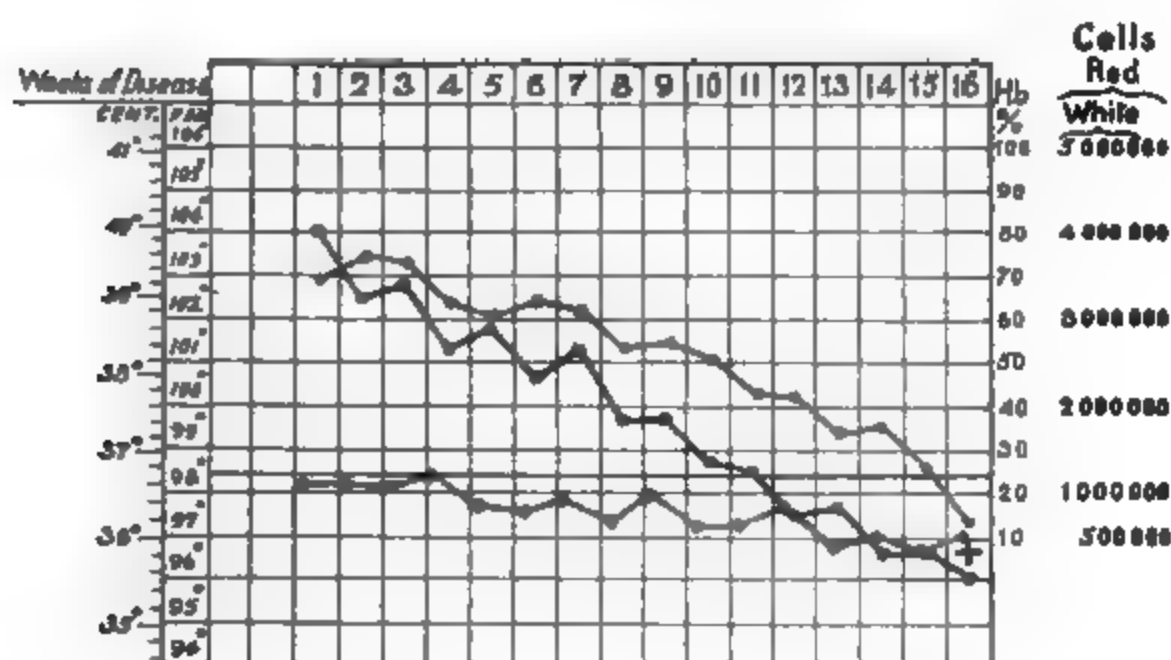


FIGURE 22.—Blood Chart. Case of Pernicious Anæmia terminating fatally. Black line indicates number of red blood corpuscles. Red line indicates hæmoglobin index. Blue line indicates number of leucocytes.

the red cells, tailed cells, and cocked-hat-shaped cells being extremely common; the blood also coagulates badly, and the red cells do not form the typical rouleaux. There are many polycytes, measuring 12-15 μ , and also microcytes measuring 4 μ . Nucleated red cells or normoblasts are almost invariably present, and often large nucleated reds or giantoblasts, and the red cells have a special staining affinity for iodine. Leucocytes are generally diminished in number, especially in cases in which the disease is progressing unfavourably. In some instances myelocytes may be found in the blood, but

¹ Since writing the above the author has seen a case in which pernicious anæmia developed and proved fatal in 10 days after confinement, and at the time the uterus was found to be gangrenous. The subject, aged 33, had, however, been anæmic for some time previous to the birth of the child.

generally in small numbers. The blood-plates are diminished, and the specific gravity is markedly lowered.

Hæmorrhages are common, mostly as small petechial hæmorrhages, subserous, and more rarely subcutaneous in position, while small hæmorrhages occur in the retinae, in connection with the meninges and elsewhere.

The *bone marrow* is usually deep red, and has an almost gelatinous appearance. It is unduly rich in cells, which are large and nucleated.

The *liver* shows fatty degeneration, and there is great excess of iron pigment in many organs of the body, including the liver, spleen, kidneys, and bone marrow; its presence is easily demonstrable by the ferrocyanide of iron reaction.

The *heart* shows marked fatty degeneration, and may be dilated, while there is in certain cases a sclerosis of the posterior columns of the *spinal cord*.

Clinical Features.—The disease comes on slowly, the face soon becoming pale and bloodless, and in a typical case the skin has a somewhat saffron or lemon-yellow colour. There is no great diminution in the amount of fat, but the pallor of lips, gums, and conjunctivæ is most pronounced. There is marked dyspnœa, almost always cardiac dilatation, and the presence of loud hæmic murmurs together with the usual *bruit de diable* in the neck, while palpitation is excited by any exertion. The pulse is feeble and rapid, and there is very commonly, in advanced cases, dyspepsia, vomiting, and not infrequently diarrhœa. A degree of pyrexia is often present, no particular reason for the rise of temperature being forthcoming. The urine contains an excess of urobilin, an evidence of the destruction of red blood corpuscles.

In the *Nervous System* one finds headache and giddiness, sleeplessness, and generally marked irritability, associated with langour and unfitness for continued mental effort. Retinal hæmorrhages are frequently seen.

Amenorrhœa is very common, as we can readily understand.

Relapses are apt to occur, and apparent improvement is too often temporary. Blood crises have been described, in which numbers of nucleated reds enter the circulation and remain for a few days. They may represent an effort at

Diagnosis.—The colour of the skin is very suggestive, while the clinical examination of the blood prevents any possible error being made.

Prognosis.—Very few cases recover, and it is doubtful whether even the few that do so are permanently cured. An increase of gigantoblasts is considered a grave feature.

Treatment.—Attention should invariably be paid to the possible causal factors, and especially to the presence of intestinal parasites, gastric disease, and oral sepsis. If the patient has been losing blood by hæmorrhoids or otherwise, promptly secure its arrest. It is a remarkable fact that anæmic patients of all kinds benefit greatly by hill air, and this should certainly be tried in cases of pernicious anæmia, together with a good and nutritious dietary.

The only drug which seems to be of genuine benefit is arsenic, and it should be given in 5-minim doses thrice daily, and gradually increased until 10 or 12 minims are being taken with each dose. It is doubtful whether iron does any good, and, in fact, the uselessness of iron has been suggested as a means of making the diagnosis certain.

Whatever our views with regard to oral sepsis and intestinal fermentation may be, it is wise to try salol or 3-naphthol, while an attempt may be made to give red bone marrow, although its efficacy is very doubtful.

So profound does the anæmia become that transfusion of defibrinated blood or of saline solution has often been resorted to, but in only a few cases does appreciable benefit result. Oxygen inhalations are worth trying. It seems extremely probable that some toxin of unknown nature is present, and as yet we have no satisfactory means of treatment for most cases of this disease.

2. Secondary Anæmia

This may be the result of (1) a loss of blood, (2) a constant drain on the system, as in Bright's disease and prolonged disordered nutrition, (3) insufficient ingestion of food or defective digestive processes; and (4) diseases or toxic conditions which interfere with the nutrition and the activity of the great vital processes.

(1) **Loss of Blood.**—A hæmorrhage of 3 to 4 lbs. weight

of blood may prove fatal. Water and salts are quickly obtained from the gastro-intestinal tract, and even the albuminous constituents of the blood-serum may rapidly recover to more or less normal proportions, but the red blood corpuscles take weeks to return to their former numbers, and the hæmoglobin index remains low.

After a severe hæmorrhage the blood undergoes certain characteristic changes. There is marked poikilocytosis, a few nucleated red blood corpuscles (normoblasts) generally appear, and, as a rule, a leucocytosis of moderate degree develops soon after the hæmorrhage, diminishing as recovery occurs. It is a grave feature to find the mononuclear leucocytes increased, and the multinuclear proportionately diminished, for it generally signifies a rapidly fatal termination.

(2) **Anæmia from constant Drain on the System as in Bright's Disease, prolonged Pus Formation, and similar Conditions.**—The drain in this case may be rather on the albuminous elements of the blood than on the red blood corpuscles themselves, but the phenomena are the same, and the reduction in number of red blood corpuscles, in amount of the hæmoglobin, together with the appearance of poikilocytosis, may exactly correspond to what obtains after a severe loss of blood. Where, however, the drain is a continuous one, the appearance of nucleated red blood corpuscles (normoblasts) is infrequent.

(3) **Anæmia due to Insufficient Food.**—Inability to swallow food, or to digest food when swallowed, may account for inanition. There may be the failure to elaborate food when absorbed, circulatory defects preventing the proper utilisation of what has been elaborated, and respiratory disease preventing the aeration of the blood, and these may all indirectly, if not directly, lead up to the same result. In such cases the plasma is believed to suffer more markedly than the red blood corpuscles.

(4) **Toxic Anæmias** are the result of certain poisons, such as lead, mercury, syphilis, or malaria, which destroy the red blood corpuscles. Probably these and similar agents act more markedly in cases in which digestive and other errors are also existent.

The **Clinical Features** of secondary anæmia, whether due to hæmorrhage or not, are much the same in all cases. We find marked debility, langour, pallor of countenance, and a

tendency to giddiness and faintness. There is inability for work, physical or mental. In the *Alimentary System*, the appetite is poor, and digestion unsatisfactory, and generally obstinate constipation is present. In the *Hæmopoietic System*, besides the changes in the corpuscular elements in the blood, the hæmoglobin index is always low. In the *Circulatory System*, the patient complains much of palpitation, and often of giddiness, which may be traced to circulatory weakness, and the extremities are apt to be cold and chilly. Hæmic murmurs are common, and include a loud systolic murmur, well heard in the mitral and pulmonary areas, and sometimes the tricuspid, associated often with a loud *bruit le diable* in the veins at the root of the neck. In severe cases a definite degree of cardiac dilatation is present. Hæmic murmurs, with the *bruit le diable*, are described under chlorosis. In the *Integumentary System*, the patient frequently complains of swelling of the legs, and especially the ankles. Certain cases show a marked diminution of subcutaneous fat, and the patients may become rapidly emaciated if the anæmia is due to any wasting disease. In the *Reproductive System*, amenorrhœa and dysmenorrhœa are commonly present. In the *Nervous System*, neuralgia is a constant cause of complaint, and headache, especially in severe cases, is apt to harass the patient. Reference has already been made to the inability for mental work, and sleeplessness is extremely common, probably the result of irritation of brain centres from a deficient supply of normal blood. In the *Locomotory System*, mention must be made of the patient's debility and the muscular weakness.

The **Diagnosis** resolves itself largely into a question of the cause of the anæmia, and not its existence, and the careful investigation of the blood helps much in deciding this matter.

The **Prognosis** depends on the possibility of removing the cause of the condition. Certain cases do well, but it is always a serious matter to find an excess of lymphocytes in the blood, while giantoblasts and macrocytes, so typical of the grave forms of pernicious anæmia, suggest a fatal termination.

Treatment.—Attempt to discover and then to relieve the cause of the condition. Should there be any alimentary error, endeavour to correct it, investigate the condition of the teeth, the stomach, the intestine, and perhaps, above all, the diet which the patient is taking. Order plenty of fresh air, easily digested and

suitable food, and, if possible, sunshine. As noted under chlorosis, the patient should be kept in bed if the heart is dilated, and until such dilatation has disappeared. The protosalts of iron have gained a well-deserved notoriety in connection with anæmia, and, in not a few cases, the addition of a small dose of arsenic is very helpful. It should not be forgotten that oxygen inhalations will greatly relieve the breathlessness and the discomfort of profound anæmia, even in cases in which satisfactory treatment is not possible.

II. LEUCOCYTHÆMIA

(LEUKÆMIA)

A DISEASE in which the white blood corpuscles greatly increase, the increase often being limited to one or more varieties of these corpuscles, and in one type myelocytes or marrow cells may be found in large numbers. There are also distinctive changes in the spleen, bone marrow, and lymphatic glands, the degree varying in different types of cases. The disease was described by the late Professor Hughes Bennett in 1845, and it is to him that we are indebted for the name leucocythæmia. From the large number of white blood corpuscles he described the disease as "suppuration of the blood."

There are two types of cases: (1) The Spleno-medullary Type, and (2) the Lymphatic Type.

1. The Spleno-Medullary Type

Etiology.—This type is more common at middle life, and apparently quite as often in males as in females. In women the climacteric is the commonest period of attack. Malaria and syphilis are possible causes, and in some families there is a hereditary history, which, however, is not very satisfactorily proved. It is doubtful whether injury alone is responsible for many cases.

Pathological Anatomy.—The spleen is reddish-brown in colour, and is not inaptly described as resembling raw beef-steak. Its weight is usually 4 or 5 lbs., but may be

anything from 2 to 18 or 20 lbs. There are generally some adhesions; on section the Malpighian bodies are invisible, the condition being a chronic hyperplasia, with thickening of the capsule. There are often numerous hæmorrhages to be seen in the cut surface, and sometimes whitish lymphoid masses scattered throughout the organ. The bone marrow presents a typical appearance resembling pus. Generally the medullary canal is expanded, there is marked hyperplasia of the marrow instead of the usual amount of fat, and on microscopic examination, numerous myelocytes, nucleated red blood corpuscles, and eosinophile leucocytes are noticeable in addition to other cells. It is not so common, in this type of the disease, to find lymphoid nodules scattered throughout the organs of the body, although it is true that this type and the lymphatic type are sometimes coexistent.

The emaciation is extreme, and dropsy is sometimes present. After death the heart and veins are distended with clots, and these have a greenish-yellow appearance. There are not infrequently hæmorrhages, sometimes petechial; retinal hæmorrhages are not uncommon.

Clinical Features.—The onset is always insidious, the enlargement of the spleen, the dyspnœa, the palpitation, and the tendency to epistaxis being often responsible for bringing the case before the notice of the physician. The spleen is painful, and especially so on palpation, the splenic notch or notches can usually be distinctly felt, and the enlarged organ extends towards the right iliac fossa. Perisplenitis is sometimes noted; and it is remarkable how the spleen may vary in size from time to time.

The blood is typical, the white cells increasing often from the normal 7, 8, or 10,000 in the cubic millimetre of blood, to 50,000, 100,000, or many hundred thousands, and the proportion to the red blood corpuscles may be increased from the normal 1 in 500 or 1000 up to 1 in 10, 1 in 5, or even equal numbers. The number of white cells varies greatly from time to time. The characteristic white cells are unquestionably the myelocytes, which are large, mononuclear cells, containing generally neutrophile, sometimes oxyphile, granules in the protoplasm. The nuclei are of large size in proportion to the cell, and of somewhat irregular shape, though generally oval. Eosinophile or oxyphile leucocytes are

increased absolutely, though not relatively. The ordinary polymorphonuclear leucocytes, while much increased in number absolutely, are often relatively diminished towards the terminal stages of the illness. Basophile leucocytes are always present, although in varying numbers, and their appearance is the more striking, because they constitute a rare cell element in the blood.

The red blood corpuscles are reduced in number, generally to about three millions in each cubic millimetre, while the presence of nucleated red cells, or normoblasts, and often also of gigantoblasts, and red cells with fragmenting nuclei, is more or less characteristic of this disease.

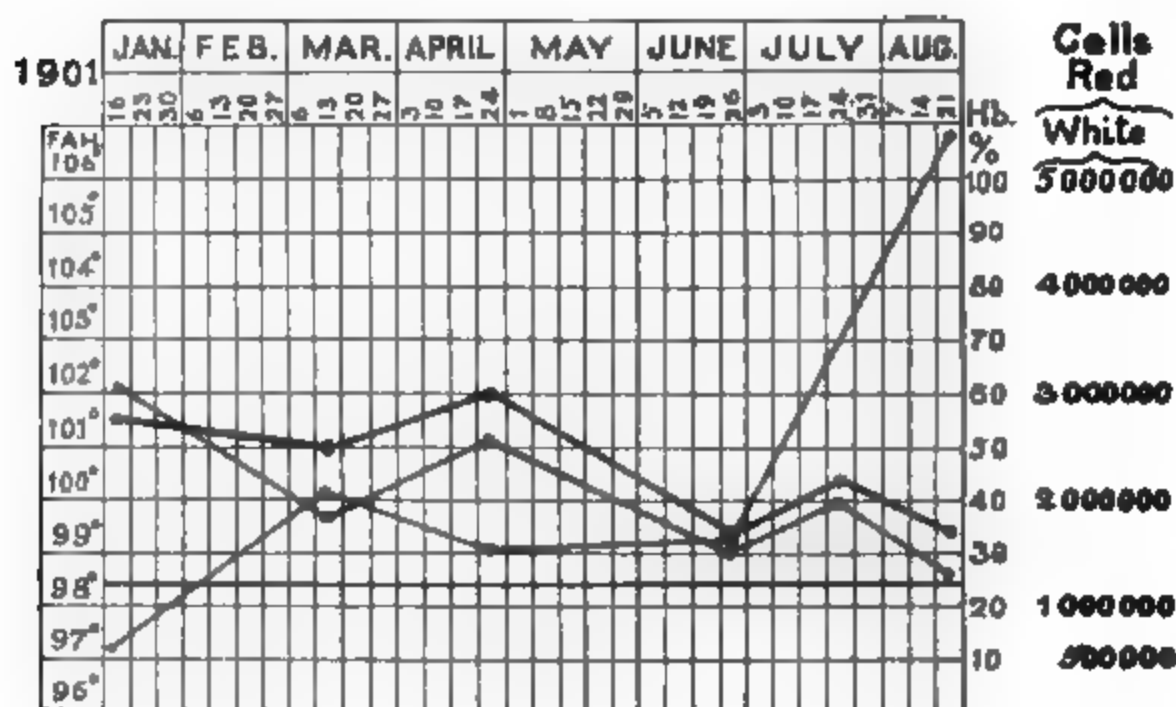


DIAGRAM 24 Blood Chart. Leucocythemia. Spleno-medullary type. Fatal event. Black line indicates number of red blood corpuscles. Red line indicates hemoglobin index. Blue line indicates number of leucocytes.

There is no special change in the blood-plates. They are certainly not reduced in number. The hemoglobin is proportionately diminished, but the blood coagulates badly owing to the presence of albumoses; and leucin, xanthin, and other bodies, due to the breaking-down of leucocytes, are found in excess in the blood. Charcot-Leyden crystals may be seen in the blood in most cases, although they only precipitate on the slide. In the *Alimentary System*, there is a tendency to nausea and vomiting, while diarrhoea is apt to be severe. Bleeding may occur from gums, stomach, or other part of the alimentary tract. In the *Circulatory System*, the phenomena are mostly associated with the anæmia. There is palpitation, a rapid and

soft pulse, and not infrequently œdema develops, although it is really of hæmopoietic origin. In the *Respiratory System*, there may be a final œdema of the lungs. In the *Integumentary System*, purpuric hæmorrhages are not uncommon, and sometimes even local gangrene. In the *Urinary System*, hæmaturia may develop, but the most important clinical feature is the presence of an excess of uric acid in the urine. In the *Nervous System*, the headache, giddiness, and tendency to syncope are all due to the anæmia. Cerebral hæmorrhage may occur, and leucocythæmic retinitis, often with hæmorrhage, interferes seriously with the patient's vision. Deafness may also be present, due to hæmorrhage.

A remarkable feature associated with the disease is the repeated occurrence of periods of pyrexia with apyretic intervals. The temperature, however, rarely exceeds 102° F.

Diagnosis.—An examination of the blood is amply sufficient in most cases, although it must be remembered that from time to time the number of leucocytes may greatly diminish, and apparently the spleen proportionately decreases in size.

Prognosis.—Life may be prolonged for a considerable time, and recovery has even occurred, but the disease is a serious one, and a guarded opinion should always be given.

Treatment.—Arsenic is *the* remedy, and it should be administered in large doses, or rather in increasing doses, care being taken to obviate its irritant effect on the alimentary tract and the onset of neuritis. The best time to administer the drug is with food, and beginning with 5 minims of Fowler's solution of arsenic, the quantity may be pushed up to 15 minims thrice daily. Otherwise, the food, clothing, rest, and the general comfort of the patient, are the only matters which call for special care.

2. The Lymphatic Type

This type is met with in young persons, and was formerly considered to be of rare occurrence. It seems probable, however, that it is much more frequent, at least in a mild form, than present statistics would indicate. There is no special etiological factor which is known to produce this form of leucocythæmia.

Pathological Anatomy.—While there may be some splenic enlargement, the chief lesion, other than in the blood, consists in enlargement of the lymphatic glands and the development of an abnormal amount of lymphoid tissue in many organs. These small lymphatic growths are well seen with the naked eye in the liver and kidneys. The bone marrow may also be replaced by lymphoid tissue.

Clinical Features.—The white blood corpuscles do not bear the large proportion to the hæmocytes that is met with in the first type, but there is proportionately a great increase of lymphocytes. The lymphocytes may be of the small or large type, and usually one type predominates in a given case. Generally speaking there are no myelocytes, and nucleated red blood corpuscles (normoblasts) are rare. It should again be stated that cases may be present in which the spleen, bone marrow, and lymphatic glands are all affected, or any one of them may show distinctive lesions. The groups of lymphatic glands most frequently involved are the cervical, axillary, inguinal, and sometimes the mesenteric. The lymphatic type is often a serious one, and death may occur in six to eight weeks.

Diagnosis.—The presence of an excess of lymphocytes in the blood, especially if associated with enlargements of certain groups of lymphatic glands, renders the diagnosis clear.

The **Prognosis** is always grave, and the **Treatment** must be on general lines. Probably arsenic should be tried, although results are not encouraging.

Chloroma

This condition is closely allied to the lymphatic type of leucocythæmia. The cases present the following characteristics:—(1) There are lymphoid deposits in the orbit, the periosteum of the skull, the temporal fossa, and other parts, causing exophthalmos and local swellings wherever the growths exist; (2) The glandular masses are bright grass-green in colour, although this somewhat startling appearance may be only noted after death, and the growths resemble in microscopic structure a lympho-sarcoma; (3) There is always profound anemia of the lymphatic type, and hæmorrhages into the skin and mucous surfaces are common; (4) There are

lymphoid deposits in the bone marrow, in the lymphatic glands, and in the skin: the bronchial and mesenteric glands are almost always involved, other glands varying in this respect; (5) The age of the patient is invariably under 40, but the condition is most common in early life, while males have presented the larger proportion of cases.

The leading clinical features are palpitation, weakness, deafness with tinnitus aurium, facial paralysis, headache, giddiness, and sometimes apoplectiform attacks, and certain of these features are directly due to the growth of lymphoid deposits.

The result is always fatal, and generally within six months.

III. LYMPHADENOMA

(HODGKIN'S DISEASE; PSEUDO-LEUKÆMIA; MALIGNANT LYMPHOMA)

A DISEASE in which the spleen and lymphatic glands undergo a peculiar hypertrophy, and in which secondary lymphatic growths develop in these and other organs.

Etiology.—The causation of this disease is still unknown—probably syphilis, tubercle, local injury, exposure, over-exertion, and cold predispose, although they cannot alone cause the condition. The disease is more common in young persons, and specially in males.

Pathological Anatomy.—The lymphatic glands are greatly enlarged, and may be either soft or hard and nodular; a relative excess of lymphocytes is associated with the cases in which the glands are soft. The spleen is enlarged, although not to the extent associated with leucocythæmia, and on section it shows numerous greyish-white lymphoid masses varying in size from a pea to a walnut. Not infrequently there is splenitis. Hæmorrhages into the spleen are not uncommon, while sometimes infarctions occur. Lymphoid nodules may develop in many organs, including the lungs, kidneys, bone marrow, and alimentary tract.

Clinical Features.—The earliest manifestation of the disease consists in gradual loss of strength, with a degree of æmia, while the glandular masses develop first in the neck,

then in the axilla and groin, and eventually in the mediastinum and in other regions. At first the glands are discrete and freely movable, while later they become matted together and often adherent to the skin. These masses rarely suppurate, but they cause serious interference with many important structures by pressure, and in this way dyspnoea, difficulty in swallowing, grave interference with arteries, veins, and nerves, may rapidly develop. There is a tendency to epistaxis, to cedema of the feet, to bronzing of the skin in certain cases, and to pains of a neuralgic nature, directly due to pressure on nerves. The tonsils, the thyroid, and the thymus are sometimes enlarged, and, as a result of the tonsillar hypertrophy, deafness may be induced by interference with the Eustachian tube. The lymphadenomatous masses differ from tubercular involvement of the glands by their tendency to push to one side important structures, such as the trachea, œsophagus, etc.

The spleen is found to be enlarged, although not to a great extent; the red cells in the blood are markedly diminished, and there may be a poikilocytosis, and even the presence of microcytes, with a proportionate diminution in hæmoglobin, and a fall in the specific gravity of the blood, but the leucocytes are not increased in number, the only change being a relative increase of lymphocytes in a proportion of cases.

There is generally a tendency to pyrexial attacks, and gastro-intestinal disorders may be associated.

The **Diagnosis** is difficult, because the disease resembles *tubercle* and also *lympho-sarcoma*. In tubercle there is always caseation and the presence of tubercle bacilli, and, as a rule, tubercular glands do not displace important structures to the same extent as lymphadenomatous growths. As regards lympho-sarcoma, there are certainly cases in which it is difficult to distinguish between Hodgkin's disease and sarcoma. One sometimes sees lymphomatous growths, piercing the bronchial or tracheal walls in a manner suggestive of sarcoma, and the difference in histological appearances between this malignant form of Hodgkin's disease and lympho-sarcoma is not very apparent.

Prognosis.—Occasionally cases improve, and marked benefit may result from treatment, but not infrequently the

disease remains incurable, and death may occur from exhaustion, or possibly as the result of pressure.

Treatment.—Arsenic is the one, if not the only important remedy. Cod-liver oil and iron are also of value, and mercurial inunction has been tried, while Clifford Allbutt recommends tungstate of soda, which proved beneficial in one case, although it failed in others. Many other remedies have been suggested, but none can be called successful.

IV. SCURVY

(SCORBUTUS)

It is a morbid condition, characterised by increasing debility, anæmia, breathlessness on the slightest exertion, and sponginess of the gums, with a tendency to hæmorrhages in various situations.

Etiology.—The disease appears to be the direct result of an insufficiency of fresh meat and vegetables, but it occurs in almost epidemic form in Russia, and is said to be endemic in certain districts, which would suggest that it might be of infective origin. On the other hand, the sanitary conditions prevailing in the parts of Russia referred to, and the standard of education of the people, leave much to be desired.

Whether the disease is due to the lack of potassium salts, or whether the absence of citrates, malates, and other organic compounds reduces the alkalinity of the blood, it is difficult to say with certainty. Such an explanation, however, appears to fit the circumstances of most cases of scurvy better than the theory that it may be due to a toxic agent in the meat or food partaken of, or a specific organism, as suggested by some Russian savants.

There seems little doubt that *predisposing* causes are ill-health, mental depression, as from confinement in prison or in besieged cities, and insanitary surroundings, as in poor and overcrowded parts of our great cities. The lack of vegetables alone is not sufficient to induce scurvy, although a modified form of the disease may result from a tea and bread dietary when associated with poverty and squalor.

Pathological Anatomy.—Hæmorrhages are specially

common and typical. They occur under the skin, under mucous and serous membranes, into muscles, under the periosteum, and sometimes into joints. There is no microscopic or other discoverable lesion of the walls of the blood-vessel to account for these hæmorrhages, and there is no leucocytosis, or any very definite change in the blood, besides what has already been mentioned. The spleen is large, congested, and soft; there may be fatty degeneration of the heart muscle, the mucosa of the gums is spongy and ulcerated, and often the teeth are shed.

Clinical Features.—Debility and languor are the earliest phenomena, and they may begin insidiously and advance slowly. Then the gums become spongy and bleed too readily, while the breath is fœtid. The patient is pale and anæmic, the teeth become loose, and hæmorrhages occur. These are petechial hæmorrhages on the legs, sometimes larger hæmorrhages into the muscles or under the periosteum, and they are often directly due to a blow or injury, the part injured becoming the site of an extravasation of blood. Coincidentally, bleeding may occur from the mouth, nose, bowel, or kidney, and œdema of the ankles is a common feature of this stage of the disease. Palpitation and dyspnœa on the slightest exertion are most troublesome.

Disturbances of the *Alimentary System* are not uncommon, especially dyspepsia, and sometimes diarrhœa. In the *Integumentary System*, the brawniness of the skin over a large hæmorrhage, even when deep-seated, is very characteristic; the petechial hæmorrhages have already been noted. In the *Urinary System*, the presence of albumin in the urine is common, but the phosphates are said in some cases to be increased, in others diminished. In the *Nervous System*, the chief symptom is the depression of spirits almost amounting to melancholia, while in the *Locomotor System* there is always a tendency, when subperiosteal hæmorrhages occur in young patients, for the shafts of the long bones or the epiphyses to be separated and to undergo necrosis, while joints may become fixed, or absolutely ankylosed, as the result of hæmorrhagic effusions. In cases which terminate fatally, extensive effusions, often hæmorrhagic, may occur into pleuræ, pericardium, etc.

Diagnosis. — When scurvy appears on shipboard or in prisons, it is common to find a number of the more weakly

individuals attacked. In towns where insanitary surroundings, combined with a diet of tea and bread and butter, and no fresh meat and little milk, are responsible, the history of the dietary may help, while purpuric hæmorrhages are associated with spongy gums in scurvy.

The **Prognosis** is favourable, if proper treatment is carried out, but when a case has gone very far downhill it may be difficult to effect a cure.

The **Treatment** should consist of plenty of fresh meat, preferably given scraped, owing to the state of the patient's teeth, and milk. Raw meat is better than cooked. The mouth must be energetically treated with antiseptic solutions, such as permanganate of potash, chlorate of potash, or carbolic acid and glycerine, and in some cases a solution of silver nitrate (10 per cent) should be painted on the gums occasionally. Give lime or lemon juice not merely as a beverage, but as a medicine 3 or 4 times a day; also plenty of fresh fruit may be eaten, while later on fresh vegetables are of great value.

Scurvy is rare on shipboard, nowadays, when fresh meat can be carried in refrigerators, and the perpetual salt junk is no longer a bit of sea life, but scurvy can be prevented if, after 10 days on salt meat, the crew of a vessel are placed on the regulation half-ounce of lime juice mixed with rum.

V. SCURVY-RICKETS

(INFANTILE SCURVY)

WHEN it is remembered that milk loses its antiscorbutic properties after it is boiled or peptonised, it is easy to understand how readily a child fed on Swiss milk, from which most of the antirachitic properties have also been removed, may develop a mixture of scurvy and rickets. Scorbutic women suckling their children are apt to induce the disease in their offspring. Many kinds of patent foods for infants contain no proper antiscorbutic elements.

A child showing the disease well developed is generally to 18 months old. It is debilitated, peevish and fretful, and particularly resents being moved or taken on the knee. All attempts at handling the limbs and especially the legs cause

great pain. There are swellings due to subperiosteal hæmorrhages on the legs and thighs, on the ribs, and often near the orbit, and the epiphyses may be separated from the bones. There is profound anæmia, often very marked alimentary disturbance, and sometimes diarrhœa, while the temperature may rise to 101° or 102° F. Sometimes the gums are spongy, but only, as a rule, after the teeth have appeared.

The **Prognosis** is good if the case is treated soon enough, and energetically.

The **Treatment** consists in giving the child *fresh* milk and, for a time at least, a teaspoonful or thereby of raw meat juice, or a little potato cream, which consists of mashed potato made up with cream or milk, several times daily. A small allowance of orange juice or fresh fruit is also beneficial.

It is important to remember what has just been stated, that boiled and peptonised milk, and many patent foods, have little or no antiscorbutic properties, and therefore, where children require to be fed on such foods, they ought daily to get a little raw meat juice, fruit juice, or other agent rich in antiscorbutic properties.

VI. PURPURA

AN affection of the blood or possibly the blood-vessels, in which hæmorrhages occur under the skin, under serous membranes, and elsewhere. They are due to toxic conditions; they are specially common in certain fevers, in connection with rheumatism, and almost any disease in which very profound anæmia or cachexia is produced.

Etiology.—Purpura is therefore not so much in itself a disease as a sign of a number of different diseases. Such hæmorrhages are most common in rheumatism, in which a special form may be met with, in pyæmia, malignant fevers, and any serious blood disease such as pernicious anæmia and leucocythæmia, while many poisons are capable of producing generalised or local hæmorrhages. Probably purpura neonatorum falls under the division of toxic purpuras. Where an individual is bitten by a deadly snake, one of the leading features is a remarkable diapedesis of red blood corpuscles into the tissues of the limb or part of the body bitten. Phosphorus,

potassium iodide, sometimes mercury, quinine, and many other drugs, may produce a mild type of purpura. Cachexia, whether the result of tuberculosis, cancer, a severe type of Bright's disease, or other condition, may, and especially in certain patients, give rise to purpuric hæmorrhages. There is a form of purpura to which the term "neurotic" has been applied, and hysterical stigmata are instances of this remarkable manifestation.

A condition due to so many varied causes may be met with at almost any age, but is certainly more common during early life.

Pathological Anatomy.—Many theories have been formulated by way of explanation for purpuric hæmorrhages. They are probably the result of some lesion of the vessel walls, or a change in the blood; it has been suggested that the cement substance between the cells in the vessel walls may be defective, and may therefore permit of transudation of red blood corpuscles. But it is probable that the larger hæmorrhages, met with in the more severe types of purpura, are due to actual rupture of blood-vessels.

Anæmia is an almost constant feature, and the results of anæmia may be noted in the different organs, while, owing probably to the excretion of blood pigment by the kidney, nephritis of a mild degree is occasionally present.

Clinical Features of the different Types

1. *Purpura Simplex.*—In this condition small red spots appear on certain parts of the body, and especially over the legs. They are very common in children and in young persons. They appear to be due in some measure to exercise, and they develop under the skin of the most dependent parts of the body, that is, the legs. The little hæmorrhages come out in crops, they are not raised above the skin, and on pressure the red spot does not disappear. The effused blood undergoes the usual pigmentary changes seen in an ordinary bruise. The patient may be slightly out of health, very generally owing to some other associated condition.


2. *Purpura Hæmorrhagica.*—This is a much more serious form, in which the patient not infrequently dies of hæmorrhage. The hæmorrhages are extensive and the mucous

membranes bleed freely. The mouth, nose, eyes, stomach, bowels, kidneys, and the female genital organs may all bleed; and not infrequently hæmorrhages are found in connection with pleuræ, pericardium, meninges, and elsewhere. The disease is more often met with in young and delicate females, and is preceded by a few days of ill-health. The patient rapidly becomes anæmic, and suffers from severe palpitation and breathlessness. The temperature is generally raised and may reach a high figure. Occasionally hæmorrhages occur into the joints, associated with much arthritic pain; vomiting is also not uncommon, and albuminuria appears in an amount out of proportion to the blood present in the urine. Many cases may be designated as mild purpura hæmorrhagica in which the hæmorrhage, although serious at the time, is not alarming, but in severe cases the prognosis is grave in the extreme.

3. *Purpura Rheumatica, or Peliosis Rheumatica or Schönlein's Disease.*—This type is specially associated with rheumatic pains. With a varying degree of pyrexia, malaise, aching pains in the legs, and often a severe sore throat, bright red spots appear on the lower part of the legs. After 2 or 3 days, another crop of spots develops with a recurrence of joint pain. The legs are often slightly swollen and tender, and the level of the spots may rise higher up the leg with each successive attack. There is not infrequently a tendency to urticarial wheals, and erythema nodosum is sometimes associated with this type of purpura. The condition is met with in patients suffering from acute or subacute rheumatism, and generally between the ages of 20 and 40.

4. *Henoch's Purpura.*—This is a form of purpura in which there are marked abdominal symptoms; vomiting, colic, and intestinal hæmorrhage being associated with purpura and joint swelling. Sometimes the affection begins with arthritis; in other cases the gastro-intestinal phenomena originate the attack. The colic pains are intense, and there are repeated attacks lasting for weeks or months with intervals of rest; sometimes albuminuria or even nephritis is present, and the purpuric spots, while most common on the extremities, may also occur on the body and face.

This type of purpura is apparently associated with rheumatism, and occurs more frequently in childhood.



Diagnosis.—The account just given of the various types of purpura has not exhausted the list of possible varieties, and nothing has been said with regard to the purpuric eruptions due to *certain drugs* such as potassium iodide; and it is necessary, in diagnosing the particular type of the affection present in any given case, carefully to exclude the probability of the affection being due to the administration of some drug. We must also investigate the condition of the blood, and the possibility of the hæmorrhage being associated with the *hæmorrhagic* or *malignant type* of *fevers*; and *scurvy* in which hæmorrhages are so common must also be considered. If we take by themselves purpura simplex and purpura hæmorrhagica, and exclude the possibility of their being due to rheumatism, they might be designated idiopathic, and the absence of any definite etiological factor would be an important point in the diagnosis of these two forms.

Prognosis.—In most cases recovery occurs, but certainly in 14 to 15 per cent of cases of purpura hæmorrhagica death is inevitable, and a case of this kind is more grave the older the patient happens to be.

Treatment.—The patient should be kept confined to bed, and in rheumatic cases the salicylate group of remedies should be ordered. Probably turpentine administered as oleum terebinthinæ in 10-20 minim capsules is one of the safest and best of remedies, while chloride of calcium in 20-grain doses, ergot, hamamelis, the mineral acids, the perchloride of iron, and lead salts, may all prove useful in different cases. The application of iced cloths, ice-bags, and sometimes pressure over bleeding points, arrests serious hæmorrhage, and in the case of Henoch's purpura, while iron and arsenic should be administered internally, an ice-bag placed over the abdomen greatly relieves the severe colic pains.

Arsenic is worth a trial in many cases, and when cure has been effected, care should be taken to prevent any excessive exercise, and to raise the standard of the patient's general health. The writer has found considerable advantage from the use of adrenalin and other forms of suprarenal extract in checking even severe cases of purpuric hæmorrhage.

VII. HÆMOPHILIA

A CONSTITUTIONAL tendency to hæmorrhage.

Etiology.—The hereditary tendency is transmitted through the female line to the male offspring, and may run through many generations. Occasionally isolated cases of the disease occur, and apparently it is associated with a peculiarly fine, smooth type of skin. The proportion of males to females affected by the condition is about 9 to 1.

As regards race, Jews probably suffer in a remarkably large proportion of cases. The condition may show itself at an early period in childhood, as for example during the rite of circumcision, but as a rule it does not appear until the end of the first year.

Pathological Anatomy.—In the case of persons who die from excessive bleeding, perhaps as the result of a trivial operation, such as the extraction of a tooth, the post-mortem changes are generally negative; but there is little doubt that the cause of the condition is associated with feebleness of the arterial and capillary walls, and is probably an embryonic error, most likely in the inner coat of the vessels. In a certain number of cases joint inflammations are met with.

Clinical Features.—(1) A cut or injury may cause excessive hæmorrhage.

(2) Hæmorrhage may occur, without previous injury, from the nose, mouth, stomach, bowels, urethra, lungs, etc.

According to Grandidier, of 334 cases of bleeding in hæmophilia—

| | | | | | |
|---------------------------------------|---|---------|---|---|------------|
| Epistaxis accounted for | . | . | . | . | 169 cases. |
| Bleeding from the mouth accounted for | . | | | . | 43 „ |
| „ | „ | stomach | „ | . | 15 „ |
| „ | „ | bowels | „ | . | 36 „ |
| „ | „ | urethra | „ | . | 16 „ |
| „ | „ | lungs | „ | . | 17 „ |

In many of these cases it was rather a capillary oozing than the rupture of one large vessel. The bleeding may last for hours or days, and, if not checked, may cause death. As may be readily supposed, the slightest injury, such as a knock or bruise, may give rise to severe bleeding, and in extreme cases

faintness may result, or a serious anæmia may follow repeated loss of blood.

Sometimes arthritis, especially of the larger joints, generally with hæmorrhages, and often involving the knees, may in time cripple the patient.

No special change has been noted in the blood.

Diagnosis.—The family history, and especially a hæmophilic history on the mother's side, is all-important. *Scurvy* and *purpura* may so far resemble this condition, but cannot well be mistaken unless in exceptionally mild cases.

Prognosis.—Death may result from hæmorrhage. Apparently the tendency may, to some extent, be outgrown.

Treatment.—Guard persons who suffer from this condition from injury of all kinds. Remember that dental and surgical operations, blistering, and even vaccination are attended by special risk. Try to brace up the general health by cold bathing, the administration of iron, cod-liver oil, and other tonics.

For the arrest of the bleeding, pressure will be found of most value. Styptics, such as tincture of the perchloride of iron, are useful, and solutions of gelatine, injected subcutaneously, have been recommended. Wright of Netley urges the use of calcium chloride in 15-grain doses thrice daily, while ergot or ergotine should not be neglected in severe cases.

A somewhat thankless task, which, however, would do more than anything else to stamp out the disease, is the effort to dissuade the daughters of hæmophilic families from marrying.

VIII. DISEASES OF THE SPLEEN

(1) Splenic Anæmia

A SOMEWHAT rare form of anæmia, characterised by enlargement of the spleen and liver, with marked diminution in the number of leucocytes, a tendency to hæmorrhages and generally pyrexial attacks.

Etiology.—It seems probable that the disease is directly due to the spleen, because removal of the spleen has in certain

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... The pigmenta-
... be in the cells of the

... external secretion from
... where the suprarenals are
... Addison's disease, the channels
... may be blocked. Possibly
... responsible for the vomiting.

... and insidious disease, with
... anemia, feeble action of the
... with marked attacks of
... and typical bronzing of the
... the hands and fingers, axilla,
... all dark, and the mucosa of
... affected. Pressure of the garter

... coloration, and irritation, such as
... a carious tooth, will determine

Moles are also often found on the
... consist in deeply pigmented spots

... rarely patches of leucoderma are
... mentation. There is much palpitation,

... yawning, due to heart weakness.
... the back and headache are common.

... augmentation of *pregnancy, pelvic disease,*
... *anemia, chronic phthisis, and malaria,*

... and that due to *argyria* (silver poisoning),
... in colour. *Arsenic*, when taken for

... may cause very marked pigmentation.
... that soap and water has removed

... at first to indicate Addison's disease.

The duration is usually a matter of months.
... with constant advance in the symptoms.

... is very unsatisfactory. *Arsenic* and
... mvaluable. Try, cautiously, some preparation

... gland. Treat vomiting with bismuth and
... diarrhea must be promptly checked with

X. TUBERCULAR DISEASE OF LYMPHATIC GLANDS

THE tubercular glands so frequently seen in the neck belong to the domain of surgery, and do not demand any description here; but the involvement of mesenteric glands requires a brief description.

Tabes Mesenterica.—This condition is often associated with tubercular ulceration of the intestine and tubercular peritonitis. The glands involved are the mesenteric and also the retroperitoneal. The disease is common in children, and the clinical picture is very marked. There is great interference with nutrition, the patient is emaciated and anæmic, but often has the most extraordinary appetite for food. The abdomen may be distended or, less frequently, flattened. Generally definite nodules can be made out on palpation, and pain, or at least discomfort, is elicited to the touch. The child suffers from colicky pains, and diarrhœa is extremely common. The clinical features of tubercular peritonitis might be repeated here, because they are characteristic of *tabes mesenterica*.

The **Prognosis** is extremely bad, although the patient often lives for a wonderfully long time; the **Treatment** must be on general lines.

XI. DISEASES OF THE THYROID GLAND

(1) GOITRE

ENLARGEMENT of the thyroid gland, which may be *Sporadic* or *Endemic*. The Sporadic form is peculiar to women. The Endemic form occurs in certain districts, as, for example, in Derbyshire in England (Derbyshire Neck), in parts of the south of Scotland, in Switzerland, and Germany, and especially amongst the inhabitants of narrow valleys in mountainous regions. The goitre varies in nature, but there is generally a great excess of colloid material, with much new formation of blood vessel and tissue. In a second type of cases, cysts form, often of large size; and in a third group there may be great vascular

dilatation, which constitutes the most obvious pathological feature.

Clinical Features.—The *Endemic* type is found in both men and women where the disease is prevalent, and persons going to a goitrous district may become affected just in the same way as the inhabitants. The explanation is doubtful; whether it is due to limestone water or to lack of sunshine during the long winter months, cannot yet be determined. The enlargement often affects one lobe alone, or one lobe more than the other, or sometimes the isthmus of the gland. In many cases the goitre grows slowly, and, so long as it is small, causes no inconvenience to the patient, but where it is large it may compress the œsophagus or the trachea, and so give rise to great distress. It is certain that sudden death is more common in goitrous patients, and therefore in their case surgical operations should not be lightly undertaken.

Prognosis.—The disease is compatible with long life, although it must be remembered that a goitrous thyroid may be associated with myxœdema. Where the gland causes pressure, the prognosis is only favourable when operative interference is possible.

Treatment.—In most instances operation should be considered, because no drug is of much value. In cases in which the gland is not active, thyroid extract should be given, and many authorities recommend the use of iodine externally.

(2) MYXŒDEMA

A constitutional disease, dependent on the loss of the function of the thyroid gland.

Etiology.—The disease may be due to complete, or almost complete, removal of the thyroid gland, or suppression of its normal function. It is present in cases of absence of thyroid, such as one finds in cretinism, while myxœdema may be associated with disease of the gland, such as goitre, and more rarely in exophthalmic goitre; and there are many conditions which appear to have a direct bearing upon myxœdema, such as syphilis, an excessive number of pregnancies, and menstrual irregularities.

Women suffer more severely than do men (six to one at

least), and there is a tendency for the disease to run in families.

Pathological Anatomy.—The thyroid gland may be absent altogether, or it may be goitrous, or the site of a tumour or cyst. In most cases there is atrophy of the secreting gland structures, and the skin is markedly thickened, the connective tissue being increased in, or near, the hair follicles and also the sweat and sebaceous glands. This accounts for what has been aptly described as a solid œdema, and also for the loss of hair, want of perspiration, and the dryness of the skin generally.

Apparently thyroid secretion is essential for the activity of brain centres; and not merely do we find marked apathy, but slight degenerative changes have been found in cerebral cells.

The pathology of the condition, although not perfectly understood, appears to be fairly clear. The removal of the thyroid, experimentally, causes a condition closely resembling myxœdema, and the administration of thyroid extract, or transplantation of a portion of thyroid, remedies the condition. It is an iodine-containing compound called iodothyron, to which the chief thyroid properties are due. It should be remembered that there are para-thyroid glands, and that if these are active, they can carry on the thyroid functions, and it is possible that in cases where removal of the thyroid has not produced myxœdema, these glands have been left behind. The para-thyroids are markedly involved in practically every disease in which the thyroid specially suffers.

Clinical Features.—The disease develops slowly, and after operation necessitating complete removal of the gland, weeks or months may elapse before myxœdema appears. The skin becomes swollen, but is firm and inelastic, which causes loss of expression, dryness of the cuticle, loss of hair, and a marked apathetic change in the demeanour of the patient. The features are coarser, the lower eyelids often have a baggy look, and there is a yellowish or waxy appearance of the face. The hands become spade-like owing to the swollen fingers; marked chilliness is usually complained of. The tongue becomes swollen, making speech difficult, and the voice is thick and monotonous, while the mental functions are slow and dull, and the memory markedly defective. Generally speaking, the pulse falls below normal, the temperature is sub-normal, and there is a definite, if not very marked, degree of

anæmia. One of the frequent peculiarities of the patient, when the disease is well developed, is excessive irritability. Tetany may follow thyroidectomy.

The patient often falls a victim to pneumonia, tubercular disease, or other complications.

Diagnosis.—The appearance of the patient, and the solid œdema of the skin, are so distinctive as to render a mistake almost impossible.

Prognosis.—The disease progresses slowly, and if untreated would eventually terminate in death.

The **Treatment** is most satisfactory. Thyroid extract should be administered in 3 to 10 grain doses thrice daily, beginning with 3 grains and increasing slowly and cautiously. The improvement is often rapid, the skin becomes softer, and all the functions greatly improve, so that, after a period of 6 or 8 weeks, it may be almost impossible to believe that it is the same patient who is being treated.

Care should be taken not to exceed the dose for which the patient is adapted, because a rise of temperature, cardiac weakness, and possible collapse, may result from any excess of thyroid extract. Once the patient is cured, the effect should be kept up by occasional small doses of thyroid extract every 2 or 3 days. Iodothyrim may be used in place of thyroid extract in proportionate doses. It is well to bear in mind that much benefit accrues from massage of the skin, and from attention to diet, as well as to the general health and comfort of our patient.

(3) CRETINISM

This disease corresponds to myxœdema, only it occurs at birth. There are two varieties: (1) *Sporadic*; (2) *Endemic*.

(1) *Sporadic Cretinism.*—In these cases there is atrophy or absence of the thyroid gland, and as a result an arrest of development both physical and mental. The disease appears to be due either to the absence or the atrophy of the thyroid, and the causes which might account for the condition are very varied, and include syphilis, alcoholism, and maternal impressions, together with delicacy and consanguinity of the parents.

(2) *Endemic Cretinism* is met with in certain districts, and especially where there are narrow valleys in which a

minimum amount of sunshine is present during the winter months. Glacier water has also been described as a possible factor; but there is undoubtedly, in addition, something in the consanguinity of parents even in these endemic cases. The disease is most prevalent in certain villages in Swiss valleys, but is also common in the valleys of the Pyrenees, Italian mountains, and some other mountainous regions. It may be associated with the development of goitrous tumours.

The **Clinical Features** of these two types of cases practically correspond. The subject of cretinism is badly developed and stunted, and the disease manifests itself during the second year of life. The face is proportionately large, as are also the hands and feet. The alæ nasi are thick, and the nose is broad, and often lacks a bridge. The tongue is large and may hang out of the mouth; the hair is thin and dry, and in older cases tends to fall out. The teeth form badly and decay at an early period; the skin has a peculiar yellow and waxy appearance, and is wrinkled; the abdomen is prominent and pendulous, and in very marked cases the patient may be unable to stand straight. The temperature is subnormal. There is another characteristic feature, namely, large pads of fat seen in the supraclavicular regions. The mind rarely develops, and imbecility is usually present. Some cretins are excessively irritable and capricious; others are more amenable to the influence of education. It is difficult to guess the age of a particular patient because growth ceases, leaving the individual stunted and dwarf-like. Life may be prolonged for many years, and death is generally due to some superadded disease.

The **Diagnosis** is easy in most cases, and it is chiefly in children, who merely show a slight degree of the disease, that there is any difficulty.

The **Prognosis** depends largely on the age at which the patient comes under treatment.

The **Treatment** consists in the administration of thyroid extract, care being taken to avoid too constant administration. It should be remembered that the cretin may grow to a considerable extent, long after the age of puberty, and if time is not given for the bones to become strengthened, they are apt to give way under the increasing weight of the body.

(4) EXOPHTHALMIC GOITRE; GRAVES' DISEASE;
BASEDOW'S DISEASE

A disease characterised by enlargement of the thyroid, prominence of the eyes or exophthalmos, and rapid action of the heart.

Etiology.—There may be a neurotic family history, but there is usually, in addition, a history of some mental or moral shock occurring in an individual below par physically. It is much more common in females.

Pathology and Pathological Anatomy.—There is increased activity of the thyroid gland, and the typical clinical features can be produced, to a certain extent, by excessive doses of thyroid extract, while in a patient with the disease the symptoms are aggravated by the administration of thyroid. The clinical features are also exactly the antithesis of what is met with in myxœdema.

There is increased development of thyroid-gland tissue, with mucinoid change in the colloid contents of the gland spaces. The thyroid is enlarged, often more markedly on one side, accessory thyroids may also be enlarged if they are present, and the thymus frequently persists. The enlarged thyroid may be soft or firm, and there is a tendency to increase of connective tissue, which may in time almost entirely replace gland tissue, thus explaining cases which terminate with myxœdematous symptoms. There are no constant changes in the cervical sympathetic or in any other nerve centre. Cardiac dilatation is very common, and may hasten a fatal result.

Clinical Features.—It may develop slowly and insidiously, or with startling rapidity. The acute cases, which are rare, show, together with the typical features, intense gastro-intestinal disturbance and often delirium.

The chief clinical features are the following:—

1. *Tachycardia*, which usually develops early, but varies greatly in degree, and is much influenced by nervousness or excitement. The pulse-rate may be only 100, or it may be 150, 200, or more per minute. There is excited action of the heart, as shown by the visible cardiac pulsation and the throbbing arteries. Palpitation is associated and may be very distressing. The heart sounds are loud, and there is

generally capillary pulsation, a wave being even seen in the peripheral veins. The heart often dilates owing to strain.

2. *Exophthalmos* develops soon after the tachycardia. Both eyes bulge, but generally one more than the other. The protrusion may be so extreme as to prevent the lids from covering the eyeballs during sleep, and the white sclerotics show above and below the iris.

Associated with the exophthalmos are the following phenomena:—(a) Von Graefe's symptom, which is the lagging behind of the upper lid when the patient is asked to look downwards, and which is due, it has been thought, to the non-striated muscle fibres of Müller in the upper lid. (b) Stellwag's sign, which consists in the retraction of the upper lid, often very pronounced, and due to spasm of the levator palpebrae muscle. Closely connected with this is the fact that the patient winks very seldom as compared with a healthy individual. (c) Moebius' sign is the difficulty which the patient experiences in converging the eyes, when asked to look at a near point. There is rarely any defect of vision, and only in exceptional cases does conjunctivitis or corneal irritation appear. The prominence of the eyeballs has been variously explained by such theories as over-filled venous sinuses, excessive amount of fat in the orbit, and contraction of the non-striated muscle fibres of Müller in the membrane covering the sphenomaxillary fissure.

3. The *enlargement of the thyroid*, as already stated, is often more marked in one lobe. The gland frequently pulsates, and a loud hum, sometimes systolic, but usually continuous, with systolic increase of sound, is generally present. A palpable thrill is felt with the hand over the thyroid in these cases.

4. A *fine tremor* is typical, and is well seen in the arms and legs.

5. *Gastro-intestinal symptoms* are not uncommon, either gastric irritability with vomiting, or diarrhoea, or both at one time, and severe attacks may occur, sometimes threatening life.

There are many other less constant clinical features, including anæmia and excessive perspiration, and, associated with this there is great reduction in electrical resistance. Pigmentation of skin, trophic changes in skin, hair, and even the nails,

slight albuminuria, sometimes glycosuria, and irritability of temper may also be present.

The first four clinical features are typical, although one or other may be absent, and the results of cardiac dilatation are often present as the disease progresses.

Diagnosis.—The four typical symptoms render recognition of the disease easy, and even when one of these is absent, there should be little room for mistake.

Prognosis.—Mild cases often recover well, while chronic cases, after a period of definite symptoms, may undergo more or less complete cure. Advanced cases are very hopeless, and death may be due to cardiac or gastro-intestinal complications.

Treatment.—In mild cases, bodily and mental rest with tonics often effects a cure. Severe cases are most unsatisfactory from the point of view of treatment. Try absolute rest with freedom from excitement in every case. Give phosphate of soda in 1 drachm doses thrice daily, and persist with it for 6 weeks or 2 months. Digitalis and strophanthus are often beneficial for cardiac complications, and iron and tonics should be administered. Antithyroidin is very disappointing. Bromides, belladonna, ergot, and many other drugs are recommended. Galvanism and faradism have been applied over the thyroid and the cervical sympathetic, but are rarely of much use. Do not operate until medical treatment, with rest, has been tried without success, and remember the high death-rate in cases operated on. The surgical procedures recommended include removal of the whole or a part of the gland, or ligature of the isthmus. Do not excise the superior cervical sympathetic ganglia, an operation, which certainly removes the exophthalmos, but fails to relieve the other symptoms. An ice-bag applied over the heart is of great benefit for the palpitation.

XII. AFFECTIONS OF THE THYMUS GLAND

THE thymus, which is apparently necessary for the child up to the end of the second year, gradually atrophies, and, in most cases, cannot be recognised by the time puberty is reached.

Persistence of the gland has been held responsible for

a number of affections, and more especially if there is also hypertrophy. Laryngismus stridulus, asthma, and even whooping-cough are amongst the conditions which have been referred to the persistence and hypertrophy of the thymus, and an enlarged thymus can certainly produce difficulty in swallowing. It is a remarkable fact, in connection with hypertrophy of the gland, that sudden death has occurred in not a few persons in whom the thymus has not merely persisted, but was greatly hypertrophied, and it is possible that a hypertrophied thymus adds to the danger of general anæsthesia, by causing pressure on the bronchial tubes or on the vagi nerves.

Recently, enlargement of the thymus has been associated with exophthalmic goitre, although the relationship between the two affections has not yet been made clear.

Tumours of the thymus, sometimes sarcomatous, and abscesses in the gland have been recorded, but they are

DISEASES OF THE CIRCULATORY SYSTEM

I. DISEASES OF THE PERICARDIUM

PERICARDITIS

AN inflammatory infection of the lining membrane of the pericardium.

Etiology.—It is rather commoner in men, and in early adult life; it is caused generally by acute rheumatism (30 - 70 per cent of all cases), occasionally by Bright's disease, and may also be the result of tuberculosis, pneumonia, pyæmia, influenza, scarlet fever, small-pox, and other infective conditions. Sometimes it is due to aortic aneurism, trauma, cancer, hydatid cyst, abscess, etc. It may occur in many different diseases and in mild degree, but the reason for its development is often obscure.

Pathological Anatomy.—There is congestion of serous surfaces of sac, and deposition of inflammatory lymph, and this is followed, in many cases, by an effusion of serous lymph. Where the infective agents are pus-forming organisms, the effusion is purulent.

There are three stages :—

1. The **PLASTIC STAGE**, to which the condition may be limited. It is the stage of exudation of fibrinous lymph. In a severe case the heart is covered with a thick, shaggy-looking layer of fibrinous lymph, and the myocardium is often affected, definite myocarditis being present. The pericarditis in mild cases is localised in area, frequently near the root of the great vessels, sometimes over the right auricle or ventricle, and some pathologists consider that "milk spots" are the result of such limited pericarditis.

Clinical Features.—There may be no symptoms at all, or, at any rate, none which point to an involvement of the pericardium. There is usually some pyrexia, with possibly an initial rigor, generally some præcordial pain with feeble, rapid, and often irregular pulse, and a varying amount of dyspnoea.

Physical Signs.—During this plastic stage *Inspection* may reveal nothing, *Palpation* possibly demonstrates distinct friction fremitus near the base of the heart, *Percussion* is often negative, while on *Auscultation* one hears a double rub. The friction murmurs change from time to time, are not synchronous with the heart sounds, and often become louder on firm pressure with the stethoscope.

The differential **Diagnosis** between friction and a double aortic murmur is easy as a rule. The fact that the double rub does not correspond in time with the heart sounds, becomes louder on pressure, and lacks the lines of conduction of the double aortic murmurs, is conclusive. Pleuritic friction, when near the pericardium, may be in part caused by the cardiac movements, and occasionally the pericardial sac itself becomes secondarily affected. This is not uncommon in tubercular disease of the lungs and in catarrhal pneumonia, and careful and repeated auscultation may be demanded before it can be decided whether or not the pericardium is involved.

2. The STAGE of EFFUSION.—In a certain number of cases the pericarditis passes from the first stage into a stage in which a serous and sometimes a purulent effusion occurs. The effusion may amount from 8 to 80 ounces. (See also Purulent and Hæmorrhagic Pericarditis.)

Clinical Features.—Pain, if present, may be greater, and there is generally a sense of oppression or distress, which is increased on pressure being exerted over the lower part of the sternum. Dyspnoea is dependent on the amount of effusion. The pressure exerted by the pericardial sac may cause various phenomena:—the pulsus paradoxus from compression of the aorta at its commencement during full inspiration, aphonia from pressure on the left recurrent laryngeal nerve, and irritating cough due to the same cause, or from pressure on the trachea or left bronchus. Lastly, the left lung may be compressed, and may suggest a pleural instead of a pericardial effusion. In a severe case, great restlessness, sleeplessness, and even delirium are generally present.

The *Physical Signs* are very definite. On *Inspection* there is præcordial bulging, and the interspaces are widened. The expansion of the left lung is obviously deficient, and there may be bulging and very generally diminished movement in the left half of the epigastric region on inspiration. On *Palpation* the apex beat is generally absent, and, if still palpable, is displaced upwards because the heart is pushed in that direction and away from the diaphragm by the increasing fluid. There may be friction fremitus at the base of the heart, but more probably, although once present, it will now have disappeared. On *Percussion* the cone-shaped area of dulness is very definite and distinctive; the apex may reach as high as the left clavicle, while the base is at the diaphragm. The apparent superficial cardiac dulness is greatly increased. On *Auscultation* the heart sounds are always weak. There may be slight friction at the base. The only sound proportionately accentuated is the 2nd in the pulmonary area.

The differential **Diagnosis** is usually easy in cases where the effusion is considerable.

It may be mistaken for (1) a *dilated heart* :—

Pericardial Effusion.

1. Impulse absent.
2. No palpable shock.
3. Cone-shaped percussion dulness, apex above.
4. Tympanitic note in axilla.
5. Sounds muffled.

Cardiac Dilatation.

- Impulse wavy.
Weak but definite shock.
Percussion dulness of enlarged heart.
No such note.
Sounds sharp and clear, although galop rhythm may be present.

Or (2) a *left pleural effusion* which, if encapsulated, may resemble the pericardial dulness. In a pleural effusion, however, the breath sounds are absent, and the heart will probably be merely displaced with distinct heart sounds.

The **Prognosis** is usually favourable in sero-fibrinous effusions, but the greater the effusion the more guarded should be the opinion given. A considerable effusion may absorb quickly, or may run a very chronic course, and adhesions are inevitable. There may be endocarditis and myocarditis present. Purulent effusions are grave.

Treatment of Plastic Pericarditis and Pericardial Effu-

sions.—For a plastic pericarditis enforce absolute rest in bed, and give in rheumatic cases one of the salicyl group combined with an alkali. Morphia (gr. $\frac{1}{8}$ - $\frac{1}{6}$) hypodermically may be required for alleviating pain. Much relief is obtained by local treatment, which may be varied according to the case. Either (1) apply an ice-bag, or (2) hot fomentations or poultices, or (3) blisters, such as cantharidis ($\frac{1}{2}$ -2 in. square), or (4) 6-8 leeches over the præcordium. In most cases poulticing or blisters are to be preferred to the ice-bag, but for a vigorously acting heart the ice-bag yields very satisfactory results.

Where an effusion is present, tapping is sometimes necessary. Paracentesis is best performed either—(a) in the 5th interspace just inside the left mid-clavicular line, or (b) in the left half of the epigastric region between the xiphi-sternum and costal margin, the needle being pushed upwards through the diaphragm. Never remove much fluid—10-20 oz. should suffice—and guard against shock by administering stimulants afterwards. Encourage absorption by appealing to the kidneys and bowels and by local counter-irritation.

In purulent cases tapping may be tried, but a freer incision should be considered. The prognosis in such cases is very bad.

Plastic pericarditis may completely disappear, but often adhesions remain, and in cases of serous effusion, should the patient survive, adhesions follow the absorption of the fluid.

In either case we may get the next or third stage of pericarditis.

3. ADHERENT PERICARDIUM.—This may result either from plastic pericarditis or pericarditis with effusion. The size of the heart depends on whether, from interference with the work of the ventricles, or from residual blood being left in the ventricles, enlargement occurs. In hearts where there are external adhesions between pericardial sac and other structures, hypertrophy is common. It is said to occur in 90 per cent of all cases of adherent pericardium not associated with a valvular lesion.

The **Clinical Features** may show evidences of dyspnoea and pain, but these are not very definite.

The *Physical signs* are often most distinctive. On *Inspection* bulging of the præcordial region may be seen, with a varying amount of systolic retraction of the left half of the diaphragm.

and, according to Skoda and other authorities, also of the chest wall at the apex of the heart. The possible development of apical systolic retraction is open to serious question. On *Palpation* a diastolic shock felt at the base of the heart is most typical; there is undulatory pulsation in cases of marked hypertrophy, and the apex of the heart is not altered in position by rolling the patient over on to the left side. *Auscultation* merely demonstrates the feeble heart sounds present. The pulse is often a faint, but typical pulsus paradoxus, especially with full respirations.

Mediastinitis may cause eventually wide-spread phenomena, both pathological and clinical; for example, peritonitis may ensue, with ascites.

PURULENT PERICARDITIS

This is common in tubercular and pyæmic cases, and may be associated with pneumonia. Pus-producing organisms may infect the pericardial sac in connection with other diseases, but the most frequent are those just mentioned. In connection with cancer, or as the result of an incised wound or infection from a neighbouring abscess, a purulent pericarditis may originate.

The clinical features are similar to, but more acute than in, sero-fibrinous effusions, and the prognosis is less favourable.

HYDRO-PERICARDIUM

Dropsical effusion into the pericardial sac.

Etiology.—Hydro-pericardium may develop in any condition which causes general dropsy: most commonly it is of cardiac or renal origin.

The **Clinical Features** in marked cases resemble pericarditis with effusion. Dyspnoea, interference with the action of the heart, and pulsus paradoxus are usually present.

The physical signs show increased præcordial dulness and feeble heart sounds, and closely correspond with those of pericarditis with effusion.

The **Diagnosis** from an inflammatory effusion depends on the other clinical features present, and on the nature of the fluid in the sac.

Prognosis.—It aids fatal termination rather than directly causes it.

The **Treatment** must be directed to treating the cause of the dropsy, and indirectly attempting to remove the fluid present, by appealing to the bowels and kidneys. Paracentesis may have to be resorted to.

HÆMO-PERICARDIUM

Blood in the pericardial sac.

Etiology.—It may result from rupture of an aneurism into the sac, from cardiac rupture, tuberculosis, cancer, scurvy, or purpura hæmorrhagica.

The **Clinical Features** of a severe case are pallor, giddiness, dyspnœa, sense of great and increasing cardiac oppression, syncope, and death. The needle is the only diagnostic agent, and cure is improbable.

Try in **Treatment** to give absolute rest to the sufferer; but this is generally a hopeless task.

PNEUMO-PERICARDIUM

Air or gas in the pericardial sac, and it is always associated with effusion.

Etiology.—It may be due to perforation of a gastric ulcer into the pericardial sac, or to injury from a wound such as may occur in sword-swallowing. Rarely the presence of the *Bacillus aerogenes capsulatus* (Welch) accounts for the condition.

The **Clinical Features** include great cyanosis, distress, and irregular cardiac action, associated with churning movements heard on auscultation, and which are due to the movements of the heart. Sleeplessness is common, and death, with delirium, most always occurs.

II. DISTURBANCES OF RATE AND RHYTHM

(1) PALPITATION

excited action of the heart, and the recognition by the patient of the forcible cardiac pulsations.

Etiology.—It may be of nervous origin, as in hysterical patients, or may follow great excitement, when patients are

specially highly strung; it is frequently met with at puberty, at the climacteric, and also in delicate girls at the menstrual epoch. In neurasthenia, palpitation is common, and it is one of the typical phenomena in exophthalmic goitre.

Amongst other causes are gout, dyspepsia, pressure of a dilated stomach on the heart, and the action of certain toxic agents in excess, such as tea, tobacco, and alcohol. Soldiers on the march often fall out owing to excessive palpitation, the result of overstrain and consequent cardiac irritability. Lastly, palpitation occurs in some forms of cardiac disease, and also in fatty degeneration of the heart.

Clinical Features.—Sometimes the palpitation is so severe as to shake the patient's bed, and the pulse may become unduly rapid (tachycardia). Often a temporary polyuria follows each attack. In gouty and dyspeptic patients there may be the tumbling heart, which is much more alarming to the patient than its significance warrants.

The **Prognosis** depends largely on the etiological factor present.

Treatment.—Avoid any known cause of the condition, and treat the dyspepsia or gout already referred to.

An ice-bag is admirable, and may be applied over the heart for hours, days, or even weeks. Brandy, ammonia, peppermint water, and similar remedies are often useful. Tincture of digitalis in 5-10 minim doses is beneficial in some cases. Occasionally valerian relieves, especially in hysteria. Sedatives, such as the bromides, are certainly worth a trial in severe cases. Divert the patient's mind from the condition; to attain this object, stretching the arms above the head while in bed and many similar devices are often helpful.

(2) TACHYCARDIA

Rapid heart's action, and is due to shortening of the cardiac diastole. The pulse-rate may be 150, 200, or even 300 to the minute; occasionally paroxysmal attacks occur with palpitation, and these are generally associated with hysteria. It is one of the typical phenomena in exophthalmic goitre. There are cases of tachycardia in which a lesion of the vagi nerves, or a lesion in the neighbourhood of the great cardiac centres in the medulla, explains the hurried action of the heart. Dyspnoea

and cardiac pain may be present, but the condition, when not too pronounced, is consistent with a long and useful life.

The **Treatment** applicable to palpitation should be tried.

(3) BRADYCARDIA

Slowness of heart's action. Many famous men have very slow pulses, and the great Napoleon had a pulse-rate of only 40 to the minute. Bradycardia is, however, met with in cases of great exhaustion, and therefore is common after a severe attack of a continued fever, and it is often present during the puerperium, in cases of gastric ulcer, and in jaundice. In uræmia and some other toxæmic conditions, a slow pulse-rate is the rule. The pulse may be slowed down in certain nervous diseases, such as apoplexy, epilepsy, melancholia, and general paralysis, and in some other diseases such as anæmia, chlorosis, and diabetes. Digitalis and many cardiac tonics act partly by slowing down the heart.

There are cases of bradycardia which are due to blocking of the auricular stimulus to contraction at the auriculo-ventricular fibres, so that only every second auricular contraction may succeed in stimulating a ventricular systole.¹

Prognosis.—A slow pulse, apart from the diseases above mentioned, generally suggests a phlegmatic constitution, and it is compatible with long life.

Treatment.—Attend to the general laws of health, and, when necessary, give tonics, and especially strychnine and diffusible stimulants.

(4) STOKES-ADAMS' SYNDROME

A slow pulse with syncopal attacks, during which the heart-rate falls still lower. This is met with in old persons,

¹ The contraction of the heart commences at the venous orifices in the auricles, and the impulse passes from auricle to ventricle by means of a small bundle of fibres originating in the septum between the two auricles, passing downwards and forwards through what is called the trigonum fibrosum, and terminating close to the root of the aorta. Should the impulse be stopped by this band of fibres, ventricular systole does not occur, and the auricular impulse is blocked. In this way the auricular contraction may not be transmitted to the ventricle, although it should be stated that ventricular systole may occur apart from any stimulus from the auricle. Very frequently in bradycardia only every second auricular systole stimulates the contraction of the ventricles, and it is probable that feeble ventricular systoles may sometimes be explained by supposing an insufficient stimulus to contraction being transmitted through these auriculo-ventricular fibres.

and is associated with arterio-sclerosis. The pulse may only total 20 per minute, and the attacks of vertigo recur several times a day.

(5) THE PULSUS PARADOXUS

In which the pulse-waves are more frequent, but less full, during inspiration. This is due to pressure of the lungs during inspiration either on the aorta, in which case both radials will show the phenomenon, or on one subclavian, when only one pulse is implicated. Pericardial effusions and adhesions may cause the paradoxical pulse to appear at both wrists, while the subclavian, generally the right, may be involved by pleuritic adhesions in tubercular affections of the lung. It must not be forgotten that a weak and especially a dilated heart may show a slight degree of the pulsus paradoxus without any of the local affections just referred to.

(6) ARRHYTHMIA

Irregular action of the heart, of which there are many varieties. Of these the following special forms deserve mention :—

(a) The **Pulsus Bigeminus and Trigeminus** are often met with in mitral disease, and may follow an excessive dose of digitalis. They consist in ventricular systoles linked together, either in pairs or in threes.

(b) The **Intermittent Pulse** consists in an occasional, very weak contraction of the left ventricle, which records no wave at the wrists. This may be habitual, and may be constant or merely occasionally developed, or it may indicate the debility following on any severe illness; in such a case exercise, temporary excitement, or a meal may make the pulse regular for a time. There are, however, other causes, such as gout, fatty degeneration of the heart, excessive use of tea or tobacco, mental shock, and sometimes heart disease, which are responsible. The intermission occurs, generally with fair regularity, every 4, 10, or 20 beats of the pulse. It may persist through life, or may, in a patient who is predisposed thereto, follow ingestion of an imprudent or too heavy meal.

The **Prognosis** depends much on the various elements in the case and their significance.

The **Treatment** consists in rest and tonics, and often the careful regulation of the dietary.

Anæsthetics are not contra-indicated by an intermittent pulse, but they should be administered with special care.

Comparatively recently the part played by extra-systoles in the production of intermittence has become recognised. Immediately after a contraction of the ventricle, there follows a phase during which the muscle is described as refractory or inexcitable. Sometimes this phase does not immediately follow contraction, and an extra-systole or second ventricular contraction occurs apart from the physiological stimulus of a preceding auricular systole. A long refractory or inexcitable phase follows, and the physiological stimulus of the next auricular systole passes unnoticed, and the inexcitable phase may not have passed off till the second auricular systole has occurred. Extra-systoles occur under the conditions mentioned above, and the prognosis is not necessarily very grave.

(c) **Fœtal Heart Sounds**.—The two normal fœtal heart sounds are equal in intensity, and the pauses are of equal length. The adult heart sounds may acquire the fœtal characteristics during a very severe illness, such as a bad attack of typhoid fever, and in the late stages of a fatal illness of any kind, when death is impending. They are also present in grave cases of cardiac dilatation. The import is always serious.

(d) The **Gallop Rhythm** is the condition sometimes met with in cases of arterio-sclerosis and chronic Bright's disease, and so forth, in which the heart sounds resemble the rhythm of a cantering horse, and are divided into three parts. Either the 1st or, more generally, the 2nd sound is reduplicated.

(e) **Delirium Cordis** implies extremely rapid, often tumultuous, and totally irregular cardiac action. It is perhaps most commonly produced by excessive doses of a poison like digitalis.

III. PAIN IN THE REGION OF THE HEART

SUCH pain may be the result of muscular rheumatism, intercostal neuralgia, costal periostitis, or pleurisy; while flatulent distension of the stomach may displace the heart, causing very definite pain. Neuralgia of the left phrenic nerve, or of the

cardiac plexus, and a neuritis of the nerves in the wall of the aorta due to inflammatory changes there, closely simulate, if they are not the actual causes of, true angina pectoris. Hysteria is responsible for very severe pain in this region, and one of the typical hyperæsthetic areas in that disease is situated just below the left breast.

ANGINA PECTORIS ; CARDIAC NEURALGIA

Paroxysms of pain in the region of the heart, dependent upon organic disease of heart, aorta, or coronary arteries.

Etiology.—It generally occurs in males and in persons who have reached adult life, and is associated with—(1) Heart conditions, such as fatty heart, hypertrophy of the heart (with arterio-sclerosis), disease of the coronary arteries, and valvular lesions, especially aortic incompetence ; (2) Vascular conditions, such as arterio-sclerosis, aortitis, and atheroma of the aorta.

It is common in gout, and the *exciting* causes of an attack are nervous shock, peripheral irritation, and over-exertion—such as straining at stool, anger, and so forth.

Pathological Anatomy.—In fatal cases the heart is relaxed and full of blood. There is present one or more of the pathological conditions indicated above.

Clinical Features.—Sudden paroxysms of pain radiate from the præcordia to the left shoulder and arm, or the left side of neck, with feeling of suffocation and impending death : less frequently the pain radiates to the right side. The attack lasts from seconds to many minutes, during which the patient is pale, with ashy-grey face, and beads of cold perspiration are seen on the forehead. The chest is held as in a vice ; the sufferer cannot move from the position in which the attack seizes him, and he generally experiences nausea afterwards. Occasionally eructations of gas and a flow of limpid urine follow an attack. Dyspnoea of an asthmatic type, with wheezing, is sometimes present. These attacks may recur frequently, or the first may prove fatal.

It is difficult to explain the nature of true angina.

1. It has been thought to be a neuralgia either of the cardiac plexus, the 4th to the 9th intercostal nerves on the left side, or the left phrenic nerve. The pain is not akin to

neuralgia elsewhere, but neuralgia involving the cardiac plexus would necessarily be unique in its peculiar severity.

2. Cramp or spasm of the left ventricle (Heberden). Such spasm would, however, be incompatible with life, and many patients have recurring attacks.

3. Over-distension of the ventricles with blood (Traube).

4. Coronary disease, either producing anæmia of part of the heart wall, and so leading to local spasm, or vasomotor spasm of the coronaries, due to the diseased arteries, may temporarily deprive a part of the heart wall of blood. A defective blood-supply to the heart wall may be just sufficient until exercise or strain increases the demand for blood, when a sudden ischæmia of the affected area of heart wall occurs, and to this, whether it results in muscular spasm or not, the anginous pain may be due.

The 1st and 4th theories are those most favoured.

Diagnosis.—Huchard distinguishes *pseudo-angina* (hysteria) from true angina.

True Angina.

1. Adult life.
2. Males generally.
3. Attacks are not periodic.
4. Awful pain.
5. Duration, seconds to minutes.
6. Often fatal.

Pseudo-angina.

- Any age.
Females generally.
Are periodic.
Less severe.
Hours.
Never fatal.

Compare also other causes of præcordial pain.

The **Prognosis** should be most cautiously given. Very rarely is permanent recovery possible, but a first attack is not usually fatal.

Treatment.—For the attack, order nitrite of amyl in 3-5 minim capsules, which should be broken in the handkerchief and inhaled, and morphia (hypodermically), or even a whiff of chloroform might be tried, in the event of the nitrite of amyl failing to afford relief. Between the attacks, give nitroglycerine (1-2 minims of a 1 per cent solution), or nitrite of soda in 2-4 grain doses; in most cases order a course of potassium iodide. Arsenic and iron are often of value.

Remember that a quiet, well-regulated life, freedom from excitement, and the prohibition of physical and mental overstrain are essential to successful treatment.

IV. ACUTE ENDOCARDITIS

INFLAMMATION of the endocardium, especially that part of it covering the valves. It may be acute or chronic, and the acute form is often described as being either (1) simple, or (2) malignant or ulcerative.

Etiology.—Endocarditis is the result of certain organisms, and in a small but unknown percentage of cases it may be due to various toxins, probably really of organismal origin. In acute rheumatism, and less acute but also rheumatic cases, in which the joints specially suffer, endocarditis commonly occurs; the simple form is more frequently the result of rheumatism than of anything else. In at least one-third of all cases of acute rheumatism, endocarditis develops; with certain fevers, and especially scarlet fever, it may occur, while tonsillitis, which is often associated with acute rheumatism, is not infrequently the precursor of inflammation of the endocardium. In pneumonia, and occasionally in phthisis, endocarditis may be due to the organisms associated with these diseases. In chorea, endocarditis may develop, although the connection between the two diseases has given rise to considerable differences of opinion.

It is common to find that, where there is disease of the valves, whether originally inflammatory or degenerative, repeated attacks of simple endocarditis occur.

In malignant endocarditis similar etiological factors may be at work, but, in addition, the organisms found on the affected valves may be those of puerperal septicæmia, pyæmia, and erysipelas, in which group of cases the endocarditis has been termed secondary in place of primary, although such a classification is objectionable. We have no absolute border-line between simple and malignant endocarditis; the simple form may assume, gradually or suddenly, malignant characteristics.

Pathological Anatomy.—1. *Simple Endocarditis.*—The inflammation commences with hyperæmia and the exudation of fibrin, leucocytes, and blood-plates upon the affected portion of the valves. In time, wart-like vegetations result, generally of small size, and usually limited to the lines of contact of sigmoid, and the auricular surfaces of auriculo-ventricular

cusps. The aortic and mitral valves are far more commonly affected than are the pulmonary and tricuspid.

This inflammatory exudation, forming a primitive vegetation, may undergo the following changes:—

(1) It may be absorbed, leaving a perfectly healthy surface behind. (?)

(2) It may organise, and become almost entirely composed of connective tissue.

(3) It may grow, and if so, it may take on ulcerative characteristics. Should this occur, portions of the vegetation may become detached, forming emboli in the blood-stream.

(4) The vegetation may disappear, but leave behind it cicatricial changes, causing a degree of sclerosis of the cusp, and perhaps interfering with the adequacy of the valve.

To what extent it is possible for a vegetation to disappear entirely, without leaving any evidence of its existence, is a point open to question. If it does occur, it is rare. It is certain that, along with thickening of cusps, contiguous edges may become adherent to each other, and this change is sometimes present in acute and also chronic cases.

2. *Malignant Endocarditis*.—In this form the vegetations tend to grow rapidly, but they also break down, and neighbouring portions of the heart or vessel wall may become infected, setting up limited areas of endocarditis or endarteritis as the case may be. Not infrequently a valve segment is perforated, or an aneurism may develop in connection with a cusp. Sometimes the ulcerative process causes an erosion through the wall of the vessel, and in one case recently the author saw a communication, by means of such an opening, between the aorta and the pulmonary artery. Myocarditis is a common result of malignant endocarditis, and is generally localised.

Embolism in different organs, and especially in the kidneys, spleen, and brain, is an inevitable sequel to ulcerative endocarditis, while it is more rare in the simple form. In the ulcerative form, where many organisms are present in connection with the vegetations, numberless capillary emboli may be found in the kidneys, spleen, and elsewhere: whereas in other cases the areas of infarction are larger in size and less numerous. The nature of the organisms in the emboli determines whether the infarcts undergo acute necrosis, forming abscesses, or not, and the

results differ greatly in different cases. Where the vegetation has originated in the right side of the heart, pulmonary infarction occurs.

The organisms most frequently found in endocarditis are, in addition to Streptococci and Staphylococci, the Pneumococcus, the Tubercle bacillus, the Gonococcus, and the Diphtheria bacillus.

Clinical Features.—1. *Acute Simple Endocarditis.*—In many patients there are no symptoms at all, but in other cases palpitation, pain, or, at least, a sense of cardiac oppression, a rise of temperature which may be somewhat trivial, and increased and irregular cardiac action, suggest that the endocardium has become involved. A bruit or murmur is not commonly heard in early cases, but slight impurity of a sound may convey important information to the experienced ear, and especially if such peculiarity of the sound alters from day to day to an appreciable extent.

Should an orifice become stenosed or incompetent, the clinical features to be described under the respective valvular lesions may be made out.

2. *Malignant or Ulcerative Endocarditis.*—Sometimes the symptoms, referable to infarctions, direct the physician's attention to the fact that a simple endocarditis has become malignant. The general features are pyrexia, generally of hectic type, and associated with sweating, often distressing dyspnoea, and increasing weakness.

There are two types frequently described: (A) The *Typhoid* or common type, in which the temperature is irregular: there is early prostration, marked delirium, somnolence, and eventually coma; diarrhoea is also a frequent characteristic. The infarctions not infrequently suggest, in this type, the true nature of the case. (B) The *Pyæmic* type is often due to puerperal conditions. The rigors, the profuse sweating, and the typical septic phenomena, with the development of jaundice, and not infrequently abscesses in joints and elsewhere, are characteristic of this variety. Petechial hæmorrhages are not uncommon.

Other types, such as *Cerebral* and *Cardiac*, have also been described. Malignant endocarditis varies greatly according to the organism present and the virulence of that organism. In certain cases the endocarditis may become quiescent, leaving

serious valvular lesions as an evidence of pre-existing disease. In other cases a rapidly fatal result ensues.

Infarction of the *kidney* causes lumbar pain, and generally blood and albumin in the urine; *splenic* infarction is characterised by pain felt over the splenic region, and sometimes by the evidences of perisplenitis; embolism of the *brain* may cause extensive paralysis, probably a hemiplegia, together with coma and sometimes delirium; *pulmonary* embolism is described in connection with chronic valvular lesions, but it generally causes a localised area of dulness with the expectoration of blood-stained sputum, and considerable pleuritic pain (see page 460).

Diagnosis.—As already indicated, the diagnosis of simple endocarditis may demand the most careful investigation. But in the malignant type, the disease assumes much graver proportions, and the evidence of cardiac auscultation generally points to the site of the disease. The rigors and hectic sweatings are peculiarly typical, and in not a few cases a definite leucocytosis is present. Occasionally the organism is obtained by culture from the blood.

Prognosis.—A cardiac lesion implies a damaged heart, but simple endocarditis may be compatible with a long and useful life, whereas the malignant form is always grave, and in certain cases proves rapidly fatal. The danger in this latter type is invariably great, even in cases in which improvement with quiescence of the disease, occurs.

Treatment.—Absolute rest in bed is imperative, and wherever there is the least suspicion that the heart has become involved, the patient should be kept absolutely at rest, and not even allowed to sit up. In rheumatic cases the salicylates should be administered, although care must be taken to avoid the depressing effect of excessive doses. In ulcerative cases undoubted benefit follows the use of antistreptococcic serum, which may be administered in doses of 2-10 c.c. either hypodermically or by the rectum, note being taken of any fall in the temperature after each dose.

Locally, an ice-bag should be applied where there is much palpitation, and Caton's treatment, by small fly-blisters applied at the 4th, 5th, and 6th left interspaces, is sometimes advantageous. The treatment of endocarditis otherwise closely resembles the treatment of ordinary valvular lesions.

CHRONIC ENDOCARDITIS

In many cases of heart disease a simple endocarditis, of very chronic type, accounts for the valvular lesion present.

Etiology.—The causes, which produce acute simple endocarditis, may also produce the chronic variety, but it seems probable that factors, such as alcohol, syphilis, overstrain, and gout, which are mainly responsible for sclerotic or degenerative changes, may also take some part in the process. In many cases of valvular disease it is absolutely impossible to differentiate between processes primarily inflammatory and those which, from the commencement, are degenerative. In every instance where a valve segment has been damaged, there is a tendency for the development of degenerative changes in the affected structure, so that calcareous deposits as well as sclerotic changes are eventually superadded.

In the great majority of cases the left side of the heart is affected, and only in a very small proportion do the valves of the right heart suffer.

Pathological Anatomy.—Thickenings are apt to occur in the neighbourhood of the corpora Arantii, and small vegetations may develop. The changes, largely sclerotic in nature, cause considerable contraction of the valve segments and thickening of their free edges. Similarly, in the auriculo-ventricular cusps, thickenings may occur which involve not merely the edge of the valve curtain, but also the chordæ tendineæ and the apices of the papillary muscles. Such changes may be associated with marked inadequacy, or comparatively slight interference with the heart's action, and a full description is given, where necessary, under the respective cardiac lesions at each orifice, so that further detail here is uncalled for.

V. VALVULAR LESIONS

RESULTS OF VALVULAR DISEASE

WHILE it is requisite to describe, under the heading of each separate valvular lesion, the results of a valvular lesion, both as regards the heart and the organism as a whole, it is prudent to consider briefly some of those which are common to all

heart lesions, and which render more intelligible the detailed consideration of each type of valvular disease.

In health a chamber, such as the left ventricle, possesses energy sufficient for its complete and adequate contraction under ordinary circumstances, but, in addition, it has a residual energy capable of bringing about complete and satisfactory contraction, even under great strain. In valvular disease, there is not merely a great diminution of this residual energy, but the energy necessary for the ventricular systole during repose may even be inadequate. This is due to several causes :—

(1) There is nearly always enlargement of certain chambers of the heart, as the result of a valvular lesion, and an enlarged chamber demands a greater amount of energy, so as to enable it to contract fully and perfectly.

(2) In most cases of valvular disease, the chamber specially affected has a double duty to perform, because the disease of a valve generally either throws on the chamber the duty of overcoming some obstruction to the onward flow of the blood in its own proper direction, or permits of abnormal regurgitation of blood through inadequate valves.

(3) In many cases, the wall of the enlarged chamber or chambers is badly supplied with blood, or is undergoing some degenerative or inflammatory change, so that, large and muscular as it may seem, its energy may be disproportionate to the work it has to do.

Valvular disease results in — (1) *Increased Backward Pressure*, and (2) *Diminished Forward Pressure*, and under these headings may be considered the immediate and remote results of any valvular lesion, while (3) *Embolism* and *Thrombosis* sometimes occur.


(1) *Increased Backward Pressure*. — If we take as an example mitral stenosis, the left auricle first becomes dilated and then hypertrophied. The lungs become engorged, and the resultant chronic venous congestion causes dyspnoea, cough with watery sputum (oedema), and not infrequently hydrothorax. The right heart next suffers; it dilates, and, in order to discharge its functions, becomes hypertrophied, and sooner or later the right auricle dilates and tricuspid incompetence results. The venæ cavæ are next engorged, and the liver is enlarged and painful, while the patient frequently develops

slight jaundice. Following upon the engorgement of the hepatic vein in the liver the portal circulation is affected, and the organs which send their blood into that vein suffer from chronic venous congestion. In the stomach we find chronic gastric catarrh with dyspepsia, and often nausea and vomiting, while hæmatemesis sometimes results from rupture of a distended venous radicle. The pancreas is also affected, and probably chronic venous congestion of that organ shows itself by inducing an intestinal dyspepsia, which is also to some extent due to chronic venous congestion of the intestine. In many cases constipation is present, should there be deficiency of the succus entericus. The spleen becomes enlarged and is occasionally painful; the peritoneum is also drained normally into the portal system, and therefore ascites develops. It should be remembered that the œsophageal veins anastomose with gastric veins at the cardiac orifice of the stomach, and that the portal circulation has a connection with the hæmorrhoidal plexus, the latter anastomosis resulting in most cases in the development of hæmorrhoids, which may cause the patient great discomfort.

Following down the inferior vena cava, we find that the kidneys send their venous blood into that vessel, and renal backward pressure causes a tendency to great diminution in the flow of urine with the presence of albumin and sometimes blood. Following the venous system still farther, we have only to note general dropsy, especially of the extremities, and cyanosis, which is specially marked in the nose, ears, lips, and elsewhere. Backward pressure in the venous radicles of the brain tends to cause sleeplessness and sometimes nocturnal delirium.

A diagrammatic representation of the points just mentioned is shown on the opposite page, and the reader will note that, for purposes of simplicity, the right and left hearts have been dissociated.

(2) *Diminished Forward Pressure.* This is more apt to occur in aortic incompetence, although wherever the left ventricle is feeble, from whatever cause, the same phenomena may be noted. These include anæmia of all organs, and specially of the brain, in connection with which it causes giddiness, headache, and sleeplessness, and perhaps also dyspnoea: in the *Circulatory* system, and in connection with the heart itself, it possibly explains the anginous pain so com-



monly noted; in the *Alimentary* system, it causes dyspepsia and feeble digestion; in all the other organs of the body, anæmia may seriously interfere with the discharge of normal functions.

(3) *Embolism and Thrombosis* are referred to elsewhere.

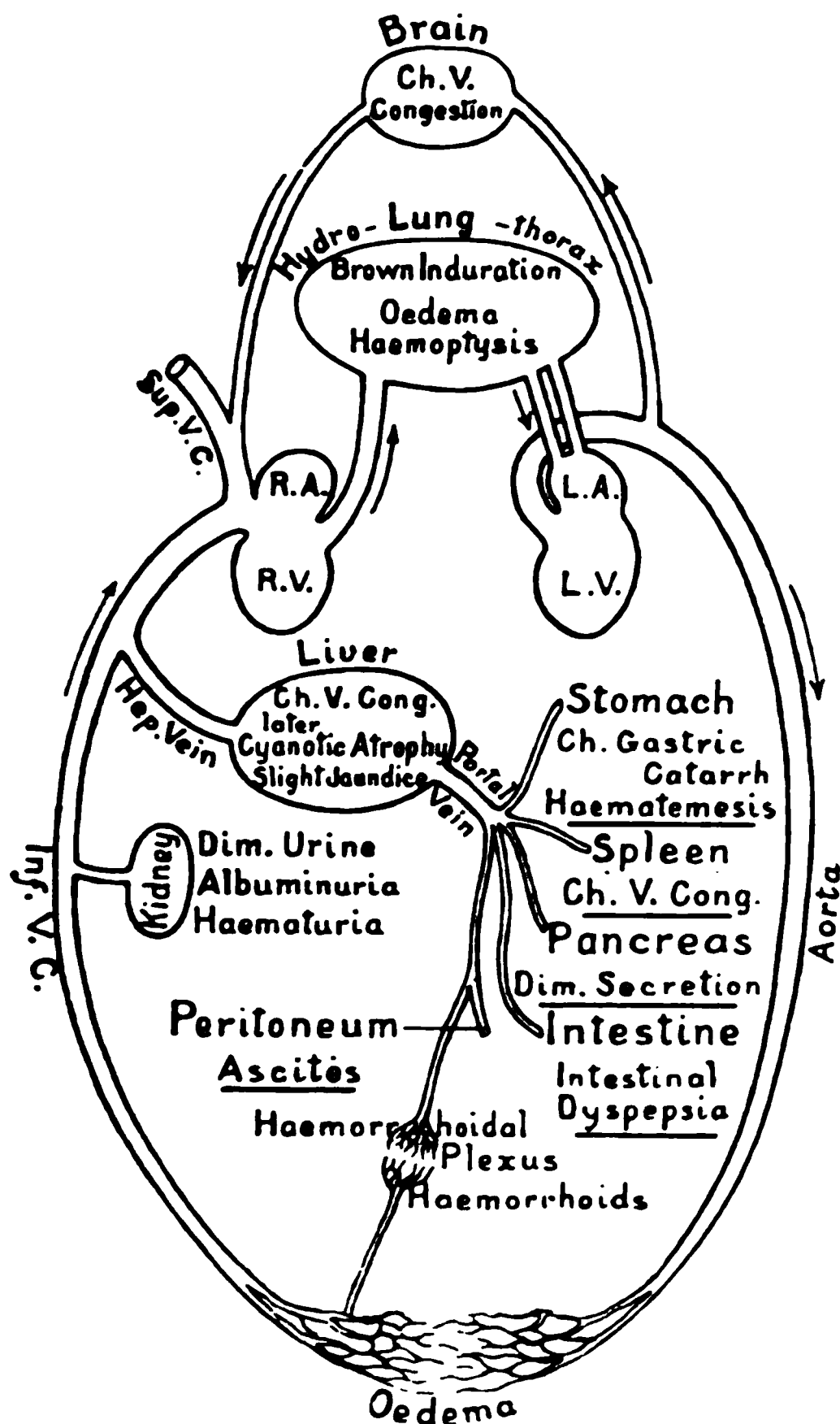


DIAGRAM 25.—Diagrammatic Representation of Backward Pressure.

It is only necessary to remind the reader that vegetations, when of ulcerative nature, give rise to emboli, and that, where the right auricle is enormously enlarged, clotting of blood is apt to occur in its appendix, so producing pulmonary embolism. Thrombosis is less commonly the result of valvular disease, but it is often directly dependent on feebleness of the circulation.

(1) Aortic Stenosis

An aortic systolic murmur may result from—

1. True aortic stenosis.
2. Dilatation of the aorta beyond the orifice, in other words, relative aortic stenosis.
3. Slight roughening of the cusps of the aortic orifice, due to vegetations or degenerative changes, and sufficient to give rise to an audible vibration in the blood-stream. It is the *first* of these conditions which we discuss here.

Etiology.—This lesion is more frequently met with in men, and during middle and later life, an age suggestive of its degenerative origin. Chronic endocarditis accounts, however, for a certain number of cases.

Pathological Anatomy.—The obstruction may be the result of vegetations or of thickening of the cusps, sometimes with adhesion of one or more of the cusps at their contiguous edges. There is mostly adhesion of cusps so as to form a funnel, opening towards the aorta, and this may be associated with little, or with a very considerable degree of, thickening. A rare form of stenosis is subvalvular in position, and is of fetal origin.

Atheroma of the aorta is very commonly present, and the coronary arteries are generally also affected in these cases. The left ventricle is hypertrophied without, at first, much dilatation, and only at a later stage does the mitral valve become incompetent, and the right heart involved owing to backward pressure. Atheroma of the coronary arteries may produce fibroid and fatty changes in the myocardium.

Clinical Features.—At first there are no symptoms, then a varying degree of malnutrition, causing giddiness and faintness due to defective cerebral blood-supply, and later other phenomena of diminished forward pressure become prominent. Anginous pain is present in some cases, and may, indeed, be an ominous development indicating progressive disease.

Physical Signs.—On *Inspection* and *Palpation* the apex beat is displaced downwards and outwards, and may be diffuse and forcible; a thrill is generally present over the aortic area, and is often very marked. The *Percussion* dulness is increased to the left, and on *Auscultation* the murmur is rough and systolic in time, with its greatest intensity over the aortic

cartilage, and it is propagated into the carotids in the neck and along the great arteries. The second sound in the aortic area is often accentuated, but varies with the condition of the cusps. The pulse is usually slow, small, and regular, and of good tension, until compensation is lost.

Diagnosis.—*Dilatation of the first part of the aortic arch* generally yields an area of dulness to the right of the sternum, and a louder and more ringing second aortic sound, and the thrill, if present at all, is rarely so obvious as it is in true aortic stenosis.

Pressure on the aorta by *tumour* should be recognised by other evidences of the new growth, while in *patent ductus arteriosus* the murmur and thrill are late systolic in time, and are loudest to the left of the pulmonary area, and the murmur is not propagated into the carotid.

Pulmonary stenosis is almost as rare a lesion as a patent ductus, and the murmur, although systolic in time, is of course not propagated along the carotid arteries.

Prognosis. — While the lesion is compatible with life and work for many years, the degree of involvement of the coronaries and the resulting degenerative change in the myocardium are the dominating factors in any given case.

(2) Aortic Incompetence

In most cases there is not merely incompetence of the aortic orifice, but, in addition, either aortic obstruction, or, at least, the presence of an aortic systolic murmur. The lesion is by no means so common as mitral stenosis, but it is very much more grave in its consequences.

Etiology and Pathological Anatomy.—Aortic incompetence occurs in two absolutely different ways:—(1) It may be the result of acute endocarditis with vegetations, not infrequently of a malignant type. This form of aortic incompetence is met with in persons generally before middle life, and is a common sequel to rheumatic endocarditis, although second in frequency to the involvement of the mitral orifice. (2) Aortic incompetence may be the result of sclerosis, especially affecting the aortic cusps, and such sclerosis is generally due to alcoholism, syphilis, or physical overstrain. It is frequently associated with atheroma, and tends to occur

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in persons who have passed middle life. These are the two most important forms. (3) Sometimes the aortic orifice is involved in a dilatation of the first part of the aortic arch, rendering the valves incompetent. (4) Aortic incompetence may also result from the rupture of a weakened cusp by strain, such as the lifting of a heavy weight, but it is rare to find such weakening of cusps apart from endocarditis. (5) The congenital union of cusps together, where, for example, there is a bicuspid condition of the aortic orifice, increases the tendency for inflammatory, and also for degenerative, processes to affect the abnormal cusp or cusps, so that malformation, although not the direct cause of aortic incompetence, may lead to its production.

Reference has been made, under aortic stenosis, to the frequent fusion at the contiguous edges of aortic cusps, and to the narrowing of the orifice which this fusion causes. It is extremely improbable that such stenosis can often occur without incompetence. Vegetations may also interfere with the adequate closure of the aortic cusps, and, even where the valve curtains have not shrunk or become thickened, one or more vegetations may not merely prevent adequate apposition of the cusps, but may even cause a serious degree of incompetence.

In the endocarditic cases of aortic incompetence, where there is much ulceration, the affected cusps may be perforated, and infective vegetations are also found on the endocardium of the heart or the intima of the aorta near the site of involvement. In the sclerotic type of aortic incompetence the valves are greatly thickened as well as retracted, and the thickening is generally best marked near their lines of attachment. There is usually atheroma of the wall of the aorta, and, in very many cases, the coronary arteries are also found to be seriously affected. The result is, that not merely do the sinuses of Valsalva fail to become distended with blood when the aortic valves are closed, but the coronary arteries, with their diminished lumen, do not convey a sufficient quantity of blood to the heart walls. The left ventricle hypertrophies in cases of marked aortic incompetence, and if the myocardium is dependent on an inadequate blood-supply, degenerative changes are inevitable. The left ventricle is sometimes so large that the heart has been termed the bovine or ox-like heart, and it may even attain the weight of 35-40 oz. As a result of the great enlargement of the chamber

of the ventricle, mitral incompetence must sooner or later supervene, leading in turn to all the phenomena of backward pressure in the lung and enlargement of the right heart.

Clinical Features.—The chief clinical features are a blowing aortic diastolic murmur propagated down the sternum, and a typical collapsing or water-hammer pulse. The patient's face is pale, and one notes the remarkable pulsation of vessels in the neck—a pulsation which seems sometimes to shake the whole body. A frequent complaint is that of pain, anginous in type, and accentuated with any slight over-exertion, while giddiness and attacks of faintness are by no means uncommon.

Physical Signs.—On *Inspection* of the præcordial region, we note a forcible and diffuse apex-beat markedly displaced downwards and outwards, and there is generally heaving, which involves the whole præcordium. *Percussion* shows marked increase of cardiac dulness to the left, and, where the right heart has begun to suffer extension, also to the right. On *Auscultation* a double aortic murmur is generally heard over the sternum. The systolic aortic bruit may be merely the result of roughening instead of actual stenosis of the orifice. The aortic diastolic is a blowing murmur, sometimes musical, and propagated down towards the xiphi-sternum, and also towards the mitral area. The point of greatest intensity is not infrequently near the base of the xiphi-sternum. Both systolic and diastolic murmurs are propagated upwards into the carotids. It is important to note the length of the diastolic murmur, because it may yield valuable information with regard to the degree of inadequacy of the orifice, a long murmur indicating less incompetence, as a rule, than a short murmur. A mitral systolic murmur is also recognised in cases in which, from dilatation of the mitral orifice, the cusps have become inadequate. Austin Flint stated that, as a result of aortic incompetence, the anterior mitral segment was pushed by the regurgitating stream of blood into closer apposition with its fellow, and that therefore a presystolic murmur might be produced by this narrowing of the mitral orifice. Much doubt has been expressed as to the correctness of this assertion; in many cases of aortic incompetence, in which a presystolic mitral murmur was also present during life, the existence of true mitral stenosis was proved on post-mortem examination.

The pulse has the typical water-hammer or collapsing character described by Corrigan, and this is readily explained by the fact that the pressure of blood in the aorta and arteries, due to the contraction of the left ventricle, rapidly falls when the blood regurgitates through the inadequate aortic orifice. The water-hammer character is well brought out when the arm is raised; associated with the sudden fall of intravascular pressure, the phenomenon of capillary pulsation can be readily seen under the nails, sometimes in the lips, and at any part of the skin when it has been reddened by friction. On ophthalmoscopic examination the retinal arteries are also seen to pulsate, a phenomenon not noted under normal conditions, and on placing a stethoscope over a large artery, such as the femoral, a double murmur may be recognised, due to the two waves passing the site of constriction and so setting up two murmurs. This is distinct from the conduction of the double murmur from the aortic orifice already noted as being audible in the carotid arteries. A sphygmogram of the pulse in aortic regurgitation shows the rapid rise, the short duration, and the rapid fall, while a predicrotic wave is often present in the down-stroke.

This description of the physical signs of aortic incompetence is typical of cases in younger persons, and which are the result of endocarditis. In older patients, there is usually a less evident water-hammer pulse, and the immediate prognosis is by no means so grave. A guide to the diagnosis of one type of aortic incompetence from the other may be obtained by comparing the time of the apex-beat of the heart with the radial pulse, when it is noted that in younger patients, in whom endocarditis is the cause of the condition, the delay in the radial pulse is very long, far longer than in health, and longer than the delay in patients whose aortic incompetence is due to sclerosis.¹

Reference has already been made to the frequency of anginous pain, and this pain, as already hinted, is probably due to the degenerative changes in connection with the aortic orifice, and perhaps specially the coronary arteries. There is not infrequently cardiac dyspnoea, and attacks of palpitation

¹ This observation of delay in time of the pulse-wave is denied by Mackenzie, but is supported by the authority of the late Dr. George W. Balfour and Sir William Broadbent.

are by no means uncommon, often indicating a very unsatisfactory condition of the myocardium of the heart.

In the *Hæmopoietic System* one notes that anæmia is often present. In the *Respiratory System*, where backward pressure has begun to be in evidence, there is cough and dyspnoea of pulmonary origin, together with œdema into the air vesicles or into the pleural sacs. In the *Urinary System*, where backward pressure is marked, some albumin is present in the urine. In the *Nervous System* there are various important symptoms of frequent occurrence: these include a tendency to faintness, and especially on rising suddenly from the prone position, and headache owing to anæmia of the brain, while sleeplessness, probably from a similar cause, frequently demands careful treatment. Many patients complain of extraordinary and harassing dreams, dreams probably largely due to irritability from anæmia of brain centres.

It is very common in ulcerative cases to find evidence of emboli either in the spleen, where they may cause pain, sometimes with perisplenitis; in the kidney, where they give rise not infrequently to hæmaturia; or in the brain, where they may cause apoplectic phenomena; but much depends on the nature of the emboli in question. In cases in which the organisms associated with the endocarditis are of great virulence, the diffusion of emboli may give rise to pyæmic phenomena, while in other cases the emboli appear to be simple. Reference has already been made to the changes which result in the area of infarction.

Diagnosis.—The distinctive murmur and pulse generally prevent any possible mistake. A *pulmonary regurgitant* murmur is better heard to the left of the sternum, and, if propagated at all, is only propagated over the right ventricle, and not towards the apex of the left ventricle. There are cases, however, in which it may be extremely difficult to eliminate the possibility of pulmonary regurgitation coexisting with aortic.

Prognosis.—There is no valvular lesion, which is so likely to cause the sudden death of the patient, but much depends on the degree of incompetence and on the condition of the myocardium. Perhaps still more depends upon the possibility of the patient leading an easy life, free from physical strain as well as mental worry, but a careful investigation should be made of the mitral orifice, the right heart, and the arteries of

the body so far as they can be examined, and valuable information may be gained from noting the position of the apex-beat.

(3) Aortic Obstruction and Incompetence

As already indicated, in considering the two preceding conditions, they are very frequently combined, and it is extremely rare to find aortic incompetence without a degree of aortic obstruction, although it should be remembered that the presence of an aortic systolic murmur is by no means necessarily due to stenosis of the aortic orifice.

In some cases the lesion begins with obstruction, in other cases it begins with inadequacy, and in both the types of aortic incompetence that have been described, there may be eventually obstruction as well as incompetence. The existence of obstruction undoubtedly increases the work of the heart, already severely strained by the incompetence of the aortic orifice, and therefore an early loss of compensation, due to the great enlargement of the left ventricle, becomes extremely probable.

The **Diagnosis** of the double lesion is not always easy. A marked degree of obstruction interferes considerably with the typical water-hammer pulse, but the existence of a very definite aortic thrill, systolic in time, greatly increases the probability of stenosis being present.

The **Prognosis** of the double lesion is grave, although the fact that obstruction is so often a sequel to incompetence, renders cases of aortic incompetence alone extremely rare.

(4) Mitral Stenosis

Narrowing of the mitral orifice.

Etiology.—In a very large proportion of cases it is the result of endocarditis. It is most common in early life, and is a disease affecting women in a larger proportion than men. Where endocarditis is present, it is the mitral valve which commonly suffers, and stenosis usually results. Rheumatism and chorea are more common in girls, and mitral stenosis is often associated with these diseases. There are, however, many cases of mitral stenosis in which it is difficult to assign a satisfactory etiological factor. It is rarely a congenital lesion.

Pathological Anatomy.—When endocarditis attacks mitral

cusps, it causes thickening and contraction, and as a rule the chordæ tendineæ suffer as well. Either (1) the disease causes a funnel-shaped orifice, due to the adjacent edges of the cusps becoming adherent and the chordæ tendineæ matted together and greatly shortened, the appearance suggesting that the papillary muscles are actually in contact with the valve curtains, while the apices of the papillary muscles become markedly fibrous; or (2) the button-hole type of stenosis may be produced, in which the disease specially involves the base of the cusps, the cusps being drawn up and puckered, and in this type the chordæ tendineæ are less affected. Both these varieties may be complicated by atheroma. The first type is believed to be commoner in young persons, the second type being associated with the disease in more advanced life, and it is hardly necessary to add that there are many cases which represent a combination of the two types together. In an advanced case, especially of the funnel-shaped type, the opening of the orifice may be reduced to an excessively small size.

As a rule in uncomplicated cases of mitral stenosis the left ventricle is not enlarged, and the changes in the other chambers are the results of backward pressure. They include dilatation and slight hypertrophy of the left auricle, dilatation and hypertrophy of the right ventricle, and often great dilatation of the right auricle. There is usually tricuspid incompetence when compensation has failed, and brown induration of the lung with marked pulmonary œdema is very common. Clots, either from the right auricle or from the tricuspid valve, not infrequently lodge in a branch of the pulmonary artery and cause pulmonary embolism; sometimes large ante-mortem thrombi may be found in both auricles, and especially in their appendices.

Clinical Features.—For many years there may be no symptoms at all. Usually dyspnœa, especially on exertion, and palpitation, are amongst the first symptoms which suggest cardiac disease. In certain cases, during an attack of rheumatic fever there is evidence of endocarditis, eventually giving rise to stenosis of the mitral orifice. Where compensation fails, great cardiac irregularity results, and the entire group of symptoms referable to backward pressure and described under mitral incompetence may be met with. Bronchitis is common,

and the degree of œdema may be similar to what is found in mitral insufficiency.

Physical Signs.—Inspection.—The apex-beat, if displaced at all, is usually displaced somewhat outwards. In cases where compensation is lost, there may be distension of the veins in the neck, and there is sometimes pulsation over the præcordial region, indicating enlargement of the right heart. *Palpation.*—A well-marked thrill, presystolic in time, is generally felt in the mitral area; it is extremely rough, and certainly closely resembles the sensation obtained if the hand is applied to a cat's head when the animal is purring. The apex-beat is very forcible, and there is sometimes a well-marked, although not so rough, diastolic thrill. There is usually epigastric pulsation, owing to the enlargement of the right ventricle. *Percussion.*—There is marked increase of cardiac dulness to the right of the sternum, and to a much less degree, as a rule, does the left cardiac border extend outwards. *Auscultation.*—A rough, short, presystolic murmur leads up to the first sound. It is probable that the so-called first sound is not in reality due to the closure and stretching of cusps, which are, too often, so rigid as to render actual occlusion of the orifice absolutely impossible, but the first sound may be due to the muscular wall of the left ventricle contracting on the blood in its chamber. A systolic murmur of mitral incompetence is frequently associated, and in marked cases there is also a diastolic murmur. The diastolic murmur is unquestionably a suction murmur. It is due to the relaxation of the left ventricle sucking blood through the stenosed mitral orifice, and hence, probably, the great length of the murmur, and also the fact that it is rarely so loud as the presystolic. There may, therefore, be a triple mitral murmur associated with mitral stenosis, and in some cases it seems as if the murmurs were continuous.

A reduplicated second sound is almost constantly associated with mitral stenosis, and it has been suggested that one part of this reduplication is actually produced at the mitral orifice, and may be, in effect, a short murmur, and it is noted that the apparently reduplicated second sound, heard in the mitral area, is often quite different from the reduplicated second heard at the base of the heart.

The pulse may be slow and regular, but when com-

pensation is lost, great irregularity generally results. It is, however, of interest to note that the irregularity is not merely one of time, but also of character of each individual pulse-wave. The force and quantity of blood in consecutive waves often show wide differences, and may constitute an important and valuable aid in diagnosis. It occasionally happens that the murmurs of mitral stenosis temporarily disappear, and an even more remarkable fact is sometimes observed, namely, that the murmurs suggesting mitral stenosis may be present, without any actual narrowing of the mitral orifice being in existence.

Diagnosis.—Should a presystolic murmur be lacking in definiteness, it is necessary to make the patient walk about, or alternately sit up and lie down once or twice in bed, so as to bring out the auscultatory signs clearly, and, on the other hand, should the heart be very irregular and rapid, rest in bed for some time may enable a diagnosis to be made.

The **Prognosis** depends much on the degree of compensation present, and also on whether tricuspid stenosis is developing or has developed. Its appearance is invariably a danger signal, but its incidence with mitral stenosis, its commonest combination, is rare.

(5) Mitral Incompetence

As the name implies, it is insufficiency of, or regurgitation through, the mitral valve.

Etiology.—1. It may be the result of atony, or dilatation of the muscular walls of the left ventricle, so that the valve segments are no longer able to cover the orifice. This condition is common in anæmia, or after any debilitating disease, while great enlargement of the left ventricle may be the result of Bright's disease, or of a valvular lesion at the aortic or other orifice.

2. Endocarditis, when involving the mitral valve, generally causes stenosis, but sometimes, with or without any very marked constriction of the orifice, the valve segments or the chordæ tendineæ may become contracted or the site of vegetations, and so prevent adequacy of the cusps. Very generally we find, secondary to endocarditis, a certain amount of calcareous deposition not infrequently at the base of one of

the cusps, sometimes even affecting the greater part of the cusp or cusps previously attacked by endocarditis, and in such a case it is extremely improbable that the rigid cusps can cover the orifice.

Pathological Anatomy.—Dilatation of the orifice referred to under the first head, and endocarditis, with or without calcareous degeneration, mentioned under the second, have already been sufficiently described when we considered the etiology, but the results of incompetency of this orifice demand a brief reference here.

There is usually dilatation with hypertrophy of the left ventricle, and if the change is permanent, and not merely temporary, the left auricle will speedily become dilated. Backward pressure in the lungs will follow next, causing brown induration, and not infrequently fairly marked atheroma of the pulmonary arteries; in a short time the right heart becomes involved. Tricuspid incompetence follows, the right auricle becomes dilated, and the general venous system is affected, this giving rise to general œdema with failure of urinary secretion. These changes imply loss of compensation, and the clinical features typical of the condition include dyspnœa, the expectoration of much watery fluid from the lung, the embarrassment of breathing due to pleural effusions, and sometimes, owing to pulmonary embolism, considerable hæmoptysis.

Restoration of compensation implies the relief of these and other symptoms, and it is generally brought about by hypertrophy enabling the more affected chambers of the heart to resume their function.

In chronic Bright's disease and in primary cirrhosis of the kidney, hypertrophy of the left ventricle may fail, in which case dilatation occurs, and the mitral orifice becomes incompetent; and in a case of adherent pericardium similar dilatation may result.

Clinical Features.—For a long period of time there may be no symptoms at all, and it must be remembered that so long as compensation is perfect, a considerable degree of mitral incompetence may be present without discomfort to the patient.

Amongst the earliest manifestations will probably be a degree of cyanosis of lips and face, shortness of breath on

any continued exertion, and a tendency to attacks of palpitation.

When compensation has failed, the following clinical features are present:—palpitation with marked cardiac irregularity, great dyspnoea with a definite degree of cyanosis; all the signs of backward pressure referable to the lung, such as watery expectoration frequently stained with blood; those referable to the stomach, such as vomiting and sickness; and those referable to the kidneys, as evidenced by the passage of scanty urine containing albumin and sometimes casts. One of the most noticeable signs of a mitral incompetence in which the patient has lost compensation, consists in the rapid development of general dropsy, beginning in dependent parts, and tending to involve the peritoneal sac, the pleuræ, and even the pericardium.

Physical Signs.—*Inspection* usually shows, in young persons, bulging of the præcordial region, and generally marked and diffuse pulsation. The apex-beat is displaced downwards and outwards, and there may be distension and pulsation in the veins of the neck. *Palpation.*—The apex-beat is generally diffuse, and is sometimes irregular when compensation has failed, but a thrill in the mitral area is distinctly rare with an uncomplicated lesion. *Percussion.*—The left border of the heart is displaced outwards, there is great increase both of relative and, as a rule, also superficial cardiac dulness, and when the right heart has become involved by the backward pressure, there is also marked increase of dulness to the right of the sternum. *Auscultation.*—The first sound is usually more or less replaced by a systolic murmur. The murmur is soft and blowing, is rarely musical, has its point of greatest intensity in the mitral area, and is propagated towards the inferior angle of the scapula, and sometimes upwards to a point just outside the pulmonary area. In rare cases it may be heard all over the chest. It should not be forgotten that a presystolic murmur will be associated in patients who have mitral stenosis, and that, when tricuspid insufficiency is superadded, a systolic murmur, different, however, in character, should be heard over the lower part of the sternum. The second sound in the pulmonary area is usually accentuated, although when compensation has failed, it is important to note how the previously accentuated sound becomes feeble. Not in-

frequently marked irregularity characterises commencing loss of compensation.

Diagnosis and Prognosis.—Recognition of the murmur is usually easy ; the difficulty is to decide upon its significance. In temporary cases, such as in anæmia, the murmur rapidly disappears with treatment, whereas if it is the result of definite organic disease, and especially endocarditis, it is improbable that much change will result in its character. The degree of enlargement of the heart, and the evidence of stenosis of the mitral orifice, where they are present, are distinctly helpful. In conclusion, temporary incompetence disappears : permanent incompetence, although alleviated by rest and treatment, does not.

(6) Tricuspid Stenosis

Constriction of the tricuspid orifice.

Etiology.—This lesion, at one time believed to be of very rare occurrence, is met with in a small proportion of cases of mitral stenosis, which it follows as a sequel, although it rarely develops to a marked degree. It may also be present as a congenital lesion, and if so, is likely to be associated with other cardiac lesions of similar origin.

Pathological Anatomy.—The affected orifice shows, but generally to a much less marked extent, the pathological conditions present in mitral stenosis. The orifice may be funnel-shaped, and there is always a certain amount of rigidity and sometimes vegetations. As regards the cardiac chambers, the right auricle is dilated and may even be slightly hypertrophied, although the muscular elements in the wall are rarely increased to the same extent as in the left auricle in mitral stenosis.

Clinical Features.—The symptoms presented by the patient are an exaggeration of those met with in mitral stenosis. There is more cyanosis and evidence of backward pressure ; the veins in the neck become engorged, and may show marked pulsation.

Physical Signs.—On *Inspection* one notes this engorgement of the cervical veins. *Palpation* may reveal a presystolic thrill felt over a wide area, and situated much to the right of the region where the mitral thrill is usually felt. *Percussion* demonstrates that the cardiac dulness is increased, particularly

to the right of the sternum, and on *Auscultation* the murmur is usually a harsh presystolic bruit with its point of greatest intensity in the tricuspid area, although it may be audible over the whole of the lower part of the sternum. It must, however, be remembered that the murmur may be absent, in which case the condition is probably never suspected during life. In many cases where tricuspid stenosis is present, it is diagnosed by the presystolic murmur, well marked in the mitral area, being audible over an unusually extensive region extending towards the right.

Very rarely is there a tricuspid diastolic murmur present.

The **Diagnosis** depends in most cases on whether, in addition to a mitral presystolic, there is a tricuspid as well. Careful auscultation, and above all the recognition of two points of maximum intensity of the presystolic murmur, are most important.

The **Prognosis** is bad, because the difficulties of the circulation are much increased owing to the tricuspid construction being as a rule superadded to the mitral lesion so generally present.

(7) Tricuspid Incompetence

Etiology.—Regurgitation through the tricuspid orifice may be the result of:—(1) acute or chronic endocarditis involving the tricuspid segments, or much more commonly (2) backward pressure, the initial lesion being often found at another orifice. In this way mitral stenosis and incompetence will lead in time to inadequacy of the tricuspid orifice; but it should also be stated that pulmonary disease greatly increases the work of the right ventricle and may lead to its dilatation. In this way emphysema and extensive fibroid disease of the lungs sometimes produce tricuspid incompetence.

Pathological Anatomy.—Where the cusps are puckered as the result of endocarditis, the condition in no way differs from what is found at the mitral orifice. Where there is dilatation, it is due to enlargement of the right ventricle, and the muscular ring surrounding the tricuspid orifice is certainly less resistant to pressure than that which aids in guarding the mitral orifice. As an inevitable sequel the right auricle dilates, and the degree of this dilatation may be extreme.

Clinical Features.—In referring to mitral lesions, a brief description has already been given, not merely of the clinical features of tricuspid incompetence, but also of its results. In slight cases there may be comparatively trifling symptoms, but where compensation is lost the physical signs are very definite, and the symptoms include dyspnoea and a varying amount of cardiac distress.

Physical Signs.—On *Inspection* the pulsation in the neck, which may be auricular or ventricular in time, is well seen in the engorged jugular veins, and it may be distinguished in the internal as well as in the external jugulars. Auricular pulsation implies that each contraction of the right auricle is registered by a backward wave into the jugular, and a systolic wave implies that the contraction of the right ventricle can transmit, directly, or possibly through the stretched tricuspid valves, a definite wave into the vein. The appearance of a double wave in the vein indicates that both the auricle and ventricle register their contractions, but where the auricle is paralysed from excessive dilatation, the auricular element disappears, leaving only the ventricular pulsation, and in certain cases no visible pulsation is seen at all, but the jugulars appear turgid and almost motionless. The waves, to which reference has just been made, must not be confused with the normal undulation often noticed in healthy jugular veins, and seen better in the external than in the internal jugulars. The venous pulsations, which have just been referred to, are not merely extremely visible, but may actually be transmitted into many of the larger venous trunks, while sometimes pulsation of the liver may be made out, synchronous with the contraction of the right ventricle. *Palpation* enables the finger to assist the eye in recognising these pulsations, and note should also be made of the occurrence of systolic epigastric pulsation, where the right ventricle is greatly enlarged. *Percussion* affords definite evidence of the enlargement of the heart to the right of the sternum, and on *Auscultation* a systolic tricuspid murmur can generally be heard over the tricuspid area and propagated over the lower part of the sternum. It is a soft, blowing murmur, often difficult to distinguish owing to the presence of a mitral systolic, but it is heard over the lower half of the sternum, and therefore differs in area from that which characterises a mitral systolic murmur.

The evidence of backward pressure in other organs is important, and the existence of the primary lesion, whether in heart or lung, greatly aids the diagnosis.

The **Prognosis** is usually somewhat grave. It is true that a mild degree of tricuspid incompetence is not serious, but when it becomes a prominent feature, it indicates that compensation has broken down or is on the point of doing so.

(8) Pulmonary Stenosis

Etiology.—It is generally congenital, but even then is extremely rare. It may be associated with a patent ductus arteriosus, or an imperfect inter-auricular or ventricular septum. In other cases it results from endocarditis, perhaps most frequently occurring in young children and associated with a severe type of endocarditis in one of the continued fevers.

Pathological Anatomy.—The common form of stenosis is due to the fusion of the cusps together to form a funnel-shaped opening, but in other cases the cusps may be thickened and retracted, with or without the presence of vegetations, while a varying degree of ulceration is sometimes present.

Clinical Features.—There is always great dyspnoea and cyanosis, which is extreme in congenital cases, but more moderate in others. It is in these congenital cases that one notes the typical clubbing of the tips of the fingers and toes, so commonly associated with the morbus cæruleus, and there is often a pulsating liver.

Physical Signs.—*Inspection* shows displacement of the apex-beat outwards rather than downwards, and there is generally epigastric pulsation. On *Palpation* a systolic thrill is almost always felt over the pulmonary area. *Percussion* demonstrates an enlargement of the right heart in most cases, and on *Auscultation* a harsh systolic pulmonary murmur may be heard, limited to its own area, but in any case not propagated into the carotids.

The **Diagnosis** is easy. An *aortic systolic murmur* is propagated into the carotids; while in a *patent ductus arteriosus* the murmur is late systolic in time, and is heard at a point in the second left intercostal space external to the pulmonary area.

The **Prognosis** is unfavourable, partly because there is a

tendency, in cases in which the lung is badly supplied with blood, to the development of pulmonary tuberculosis.

(9) Pulmonary Incompetence

Etiology.—This may be the result of conditions similar to those described under aortic incompetence, but sclerosis of the pulmonary cusps is very rare. On the other hand, the pulmonary orifice can be stretched in such a way as to render the cusps inadequate, and therefore, in conditions in which there is severe backward pressure, pulmonary incompetence may occur temporarily. Endocarditis of the pulmonary orifice is rare, but may develop in connection with acute rheumatism or one of the infective fevers.

Pathological Anatomy.—The cusps may be thickened, twisted, and sometimes adherent, so as to give rise to stenosis, and not infrequently there are vegetations, often associated with ulceration. It should, however, be stated that in cases in which the incompetence is the result of over-distension, the valve cusps may be perfectly normal, and after death the orifice may have regained its normal size. A permanent pulmonary incompetence results in dilatation with hypertrophy of the right ventricle, and when the tricuspid valve becomes in turn incompetent, dilatation of the right auricle ensues.

Clinical Features.—It is very difficult to give a satisfactory description of the results of pulmonary incompetence. Where it is of organic origin, it will certainly cause marked dyspnoea, cyanosis, and other evidences of an extreme degree of backward pressure.

Physical Signs.—On *Inspection* the veins in the neck are seen to be distended and to pulsate. On *Palpation* the apex-beat is found to be displaced to the left, and there is epigastric pulsation due to the enlarged right ventricle. The *Percussion* dulness is increased both to the right and to the left, and on *Auscultation* a murmur, generally soft and blowing, occasionally harsh and rasping, is heard in the pulmonary area. It is diastolic in tone, its point of greatest intensity is in the second left space close to the sternum, and its line of propagation is downwards towards the right ventricle. It is important to note whether the second sound, which ought to be produced in part

at the pulmonary orifice, is completely replaced by the murmur, or is still audible.

The **Diagnosis** lies between *aortic incompetence* and pulmonary incompetence. In aortic incompetence, the murmur is propagated towards the apex of the heart, and in most cases a double aortic murmur may be heard in the carotids, whereas a pulmonary diastolic murmur is only propagated over the right ventricle, and is not, even when associated with a pulmonary systolic murmur, propagated into the carotids. The water-hammer pulse and capillary pulsation are present only in aortic incompetence. The chief difficulty arises in cases in which both lesions are coexistent.

The **Prognosis** is bad in all cases, and especially where the lesion is of permanent nature.

TREATMENT OF HEART DISEASE

A case of heart disease may be divided into three stages :—

1. The stage before compensation is lost, and when very probably the patient does not come before the physician.
2. The stage when compensation is lost, a stage which may recur again and again during the life-history of any given cardiac case.
3. The stage when compensation has been regained.

Stage 1. During the first stage, should the cardiac condition be recognised, do not order any cardiac tonic, but rather endeavour to regulate the life and work of the individual so as to preserve compensation. Give strict directions as to moderation with regard to food, drink, and tobacco; in most cases forbid either too hot or excessively cold baths, and especially veto Turkish or Russian baths. Counsel the patient to avoid excitement, and remember the risk of sexual intercourse in serious cases of aortic incompetence, even at an early period in the disease. Should any drugs be ordered at all, give iron, arsenic, and general tonics.

Stage 2. When the second stage is reached, the first thing is to place the patient absolutely at rest in bed, and so aid the heart to regain compensation, if possible, without having to resort to any direct cardiac tonic. Should a cardiac tonic, however, be unavoidable, we may prescribe digitalis in the form of tincture (℥ 5-15), or the infusion (℥ 2-4). Digitalis

acts powerfully on cardiac cases, causing a fuller contraction of the ventricles, and thus strengthening the action of a weak heart, and rendering it more regular, and, at the same time, slower. It increases the flow of urine, and of its glucosides digitalin and digitoxin are powerful vasomotor stimulants, while digitonin, which is believed to be present in larger amount in the infusion, is a valuable vasodilator agent, acting like saponin. There is much difference of opinion as to the reputed cumulative properties of digitalis, but certainly it is wise to intermit the drug for a few days after its continuous administration for say 3 or 4 weeks. Like strophanthus, digitalis is apt to disagree with the patient's stomach, and may induce sickness, and it should be combined, as a rule, in such cases with a carminative. If given in excess, digitalis causes great rapidity and irregularity of cardiac action, and tends to arrest the renal functions.

Strophanthus is often preferable to digitalis, and especially where the vasoconstrictor action is not desired. It is better borne by the stomach, but should invariably, in cases where there is any tendency to irritability, be given in combination with a carminative, such as tincture of capsicum (℞ 1-5). Tincture of strophanthus is administered in doses of 5-15 minims; and although it is desirable to watch the effect with care, even when doses of only 10 minims thrice daily are being administered, it is, nevertheless, a drug which may be pushed in certain cases with great advantage. Strophanthin is sometimes preferred in doses of $\frac{1}{300}$ to $\frac{1}{100}$ of a grain.

Caffeine citrate is of special value after one or other of the preceding drugs has been administered, and when, perhaps owing to dyspepsia or other cause, it has been temporarily interrupted. The dose of caffeine citrate varies from 5-30 grains.

Indirect relief is afforded to the heart by (1) venesection, by appealing to the (2) bowels, (3) kidneys, and more rarely (4) to the skin, (5) by opening up the vascular channels, by the administration of one of the nitrite group of remedies, and (6) by tapping dropsical effusions where they have become excessive.

(1) Bleeding is specially serviceable in extreme cases of backward pressure, with engorgement of the right heart. The median-basilic vein should be selected for the operation, and

20-30 ounces of blood removed. Follow the operation with stimulants or cardiac tonics.

(2) Depletion by the bowels is carried out by the administration of hydragogue cathartics, such as magnesium sulphate ($3\frac{1}{2}$ -1), given as a concentrated solution in the morning before breakfast, or a compound jalap powder (grs. 30-50).

(3) Depletion by the kidneys is aided by such diuretics as spirit of nitrous ether in drachm doses every 4 or 6 hours, or by diuretin in 10-15 grain doses.

(4) Depletion by the skin is rarely safe practice, and diaphoretics, other than the simple liquor ammonii acetatis, should not be given.

(5) For dilating vessels $\frac{1}{100}$ th of a grain of nitroglycerine, generally administered in a solution (liquor trinitrini), is one of the best agents.

(6) The removal of the effusions from pleura, peritoneal sac, and the subcutaneous tissue of dependent parts of the body, often gives great relief to the heart. Potain's aspirator is generally used for tapping the pleura and sometimes for an ascitic effusion, while Southey's tubes are generally employed for the abdomen, and almost invariably for draining fluid from the legs.

A very important matter in the treatment of cases of heart disease, is the proper quantity of fluids which should be allowed to the patient. A dry diet, by which is meant the limitation to 20 or 30 oz. of fluids a day, diminishes œdema, and throws less strain on an overworked heart.

There are also *Special Symptoms* which demand treatment. *Dyspnœa* is relieved by resting the patient in bed, by tapping the pleura, by giving such stimulants as equal parts of the spirits of ammonia, ether, and chloroform in drachm doses, by cupping the bases of the lungs, and by oxygen inhalations. Paroxysms of dyspnœa sometimes call for morphia. *Palpitation* should be treated by applying an ice-bag over the heart. *Dyspepsia* and *vomiting* require most careful dieting, and dilute hydrocyanic acid (m 3-5) and subnitrate of bismuth (gr. 10-30) are serviceable, while a mustard leaf or poultice applied over the region of the stomach is often of great value in allaying intractable vomiting; effervescing alkaline drinks, such as potash or soda water, are also good. *Cough* is relieved by

such stimulants as spirit of chloroform or alcohol, and *sleeplessness* may require paraldehyde (m 30-90), or even morphia (gr. $\frac{1}{8}$ - $\frac{1}{3}$) hypodermically.

Hæmoptysis is beneficial when not excessive, but, unfortunately, it begets fear, and then a mineral acid, with the liquor morphinæ (m 10-15), should be given to soothe the patient.

A case of *aortic incompetence* requires special directions; here such a cardiac tonic as digitalis, by lengthening the ventricular diastole, is said by some authorities to be hurtful and even dangerous; but given with care it will prove invaluable where compensation has failed. Opium is frequently of great benefit, and this drug, together with potassium iodide, should invariably be accorded a fair trial, more especially in cases where anginous pain is present. Opium relieves the sleeplessness, dyspnœa, and cardiac asthma which are often present in this form of heart disease.

In a case of long-standing heart disease, whether valvular in origin or due to cardiac dilatation resulting from degenerative or fatty changes in the heart muscles, a time comes when rest and drugs are insufficient. In certain of these cases cautiously increasing the work of the heart seems to be of special value in toning up the diseased organ.

The writer has carried this out in the following way. The patient, after complete rest in bed and the usual cardiac tonics and other treatment, is subjected to (1) massage for 1 to 3 or more weeks; (2) passive exercises or movements of the limbs for a similar period; to be succeeded by (3) resistance exercises, more or less following the prescribed method of Dr. Schott of Nauheim; and then, lastly, (4) he is allowed to get up and walk for half an hour or thereby, and gradually day by day increases the personal effort.

The results of this treatment are excellent where the heart is still capable of improvement, and the consequent diminution in the size of the dilated chambers is extremely striking, while the improvement as regards the patient's general well-being is manifested by increased appetite, by better sleep at night, and by greater cheerfulness. Oertel's hill-climbing exercises are more applicable for cases of fatty infiltration of the heart.

The bath system of treatment, as carried out at Nauheim, deserves a few words of description:—

(1) The patients begin with baths at a temperature of about 95° F., the bath containing 1 lb. of sodium chloride, which is gradually increased to 3 lbs., and 1½ oz. of calcium chloride gradually increased until 4½ oz. are present. The bath is 10 gallons in capacity, and the patient remains for 5 to 10 minutes in the water, gradually extending the duration of the bath until 20 minutes is reached, while the temperature is lowered to 84° F. These baths are taken on alternate days, or 1 day out of 3, and a period of rest in the recumbent posture follows each bath. In two weeks the patients are ready for—

(2) The effervescing baths, which contain the full-strength solution of the salts mentioned above, and to which are added 2 oz. of sodium carbonate, gradually increased to 8 oz., and 3 oz. of hydrochloric acid, gradually increased to 12 oz.

(3) Sometimes an undercurrent douche is used with the effervescing baths. The baths may be prepared artificially, as indicated by the quantities mentioned above, the solution of salts and of carbonic acid gas closely imitating the natural mineral waters of Nauheim and the strength in which these are used for treatment.

Stage 3. When the stage of restored compensation is reached, the duty of the physician is directed to preserving it; and, as suggested under stage 1, the diet, life, and work of the patient should be carefully arranged and regulated. Arsenic is a useful drug in many cases during this period, because it conserves the cardiac energy.

VI. CARDIAC HYPERTROPHY

It is doubtful whether cardiac hypertrophy ever occurs apart, altogether, from dilatation, and it seems most probable that although the hypertrophy may be the most prominent feature, dilatation invariably accompanies it.

Etiology.—A chamber of the heart hypertrophies when extra work has been gradually thrown upon it; wherever dilatation has occurred, hypertrophy is the process by which compensation is restored. Hypertrophy is the result of excessive stimulation, as by over-exercise, excitement, or the action

of certain toxic agents in harmful amount, such as digitalis, alcohol, tea, etc. The *Left Ventricle* hypertrophies in Bright's disease, in which the arterial resistance becomes greatly increased, and also as the result of aortic and, to a less extent, mitral lesions, which throw greater work on that chamber. The *Right Ventricle* hypertrophies where backward pressure, however produced, renders the work of the chamber greater, and similarly the *Left*, and, to a much less marked degree, the *Right Auricle*, may hypertrophy where the mitral or tricuspid orifices respectively are narrowed, thereby increasing the effort necessary to empty the chamber. Pericardial adhesions may cause increase in size of the heart as a whole.

Pathological Anatomy.—Although hypertrophy has been specially referred to under the heading of etiology, as already stated, it is always accompanied by more or less dilatation, and with the exception of the left ventricle the dilatation is a more prominent characteristic than the hypertrophy. The wall of a hypertrophied left ventricle increases in thickness, the columnæ carneæ greatly increase in size, and in the auricles the muscoli pectinati become much thicker. The capacity of the chamber is also increased, although this may be only slightly so in the case of the left ventricle.

Clinical Features.—Where hypertrophy predominates in the *Left Ventricle*, as in chronic Bright's disease, marked palpitation results, with, not infrequently, considerable headache and general discomfort.

The *Physical Signs* show enlargement of the left side of the heart, and the displacement of the apex downwards and outwards, while the first sound in the mitral area has a peculiarly booming character. The pulse is often full and strong.

Hypertrophy of the *Right Ventricle* is evidenced by epigastric pulsation, extension of cardiac dulness to the right of the sternum, and the other phenomena due to the primary cause of the condition.

Hypertrophy of the *Auricles* gives rise to no special features.

The **Diagnosis** is rarely difficult so far as the left ventricle is concerned.

The **Prognosis** depends largely on the possibility of maintaining compensation. The greater the amount of

coincident dilatation the more apt is the enlarged heart to cause trouble.

The **Treatment** already described under palpitation yields the best results. Where there is no valvular cause for the hypertrophy, a quiet, well-regulated life, together with the administration of sedatives, and especially tincture of aconite, will often yield beneficial results, while the patient should be discouraged from taking an undue interest in his cardiac condition.

Hypertrophy, where it merely compensates for the damage done by a valvular lesion, does not call for treatment.

VII. CARDIAC DILATATION WITH COMPENSATORY HYPERTROPHY

THIS is simply what obtains in a case of valvular or cardiac disease in which compensation has already been so far lost and is again restored. It is unnecessary to describe here the condition of the heart common to most forms of ordinary valvular disease, and the clinical features in a marked case are those of a patient whose compensation is barely maintained. There is palpitation, pain, breathlessness on exertion, and a tendency to cardiac failure after any excess of effort above the compensatory powers of the heart. The physical signs show enlargement of the chamber or chambers. In the case of the left side of the heart, percussion dulness demonstrates an extension both downwards and to the left, and where the right side of the heart is affected, the border is markedly displaced outwards to the right. The engorged veins, and the irregularity in both heart and pulse, yield significant signs, and the treatment consists in an attempt to diminish the dilatation and to increase the residual energy of the heart.

VIII. CARDIAC DILATATION

By this is meant enlargement of a chamber or chambers of the heart without proportionate and compensatory increase in their muscular walls.

Cardiac dilatation is a common result of any acute febrile process, as well as of profound anæmia, in which the heart wall

is poorly supplied with blood. It may also occur in diphtheria, or as the result of some toxin acting on the heart wall, or possibly the cardiac centres; tobacco is an excellent example of an agent of this kind. Further, dilatation of the heart is secondary to valvular lesions, myocardial degeneration, disease of the coronary arteries, Bright's disease, and many other conditions.

Overstrain is a common exciting cause of dilatation, and if the heart is damaged from valvular disease, malnutrition of the heart wall, the action of a toxin or an unsatisfactory blood-supply, it acts with greater effect, and a smaller amount of effort produces a serious degree of distension.

It is probable that a dilated chamber of the heart does not become completely emptied during its systole, and the residual blood tends by degrees to increase the dilatation. Very rarely rupture of a dilated heart occurs. As a general rule the muscle fibres are over-stretched, and the chamber does not recover its compensation until hypertrophy occurs.

The **Clinical Features** are those of loss of compensation, with the phenomena of both increased backward pressure and diminished forward pressure markedly present. There is much cardiac distress, often an irritative type of palpitation, and faintness is induced by the slightest effort. The digestion is poor, and constipation, more rarely diarrhoea, adds to the troubles of the patient. Sleeplessness is frequently complained of, and general dropsy soon becomes marked.

The Physical Signs are definite. There is not merely enlargement of the affected chambers of the heart, or the heart as a whole, but the apex-beat is feeble and often irregular, while on *Auscultation* the sounds of the heart are loud, but lack strength, and have been well described by the term "flapping." Further, the gallop rhythm is very typical, and in many cases Cheyne-Stokes breathing is noted during sleep. The engorged veins in the neck which often pulsate, the enlargement of the liver, and the many other symptoms and physical signs referred to under increased backward pressure, may be manifested.

The **Diagnosis** is comparatively easy, although sometimes a *pericardial effusion* closely simulates a dilated heart (page 334).

The **Prognosis** depends on the possibility of the restoration

of compensation, and therefore, to a considerable extent, on the primary cause of the dilatation. In older patients, or in long-standing cases of heart disease, dilatation is grave.

Treatment.—While everything might be repeated which has already been stated under the treatment of valvular lesions, the following points are worthy of special attention:—

1. Endeavour by rest to relieve the heart and to reduce the dilatation.

2. Remember that diuretics and purgatives will indirectly afford relief to the overburdened heart.

3. Give cardiac tonics and diffusible stimulants, although they should be administered with care.

4. Endeavour to relieve symptoms as they occur; and

5. Much may be done in the way of toning up the heart by massage and other methods described on page 372, although obviously there is a limit to the possibility of improvement in many cases.

IX. DISEASES OF THE MYOCARDIUM

(1) MYOCARDITIS

AN acute or chronic inflammation of the myocardium.

Etiology.—An acute diffuse myocarditis is sometimes a sequel to diphtheria, scarlatina, and more rarely other fevers, but generally, whether acute or chronic, it is secondary to an endo- or pericarditis, and is more or less localised. It may also occur in pyæmia from the infective organisms entering the coronary circulation.

Pathological Anatomy.—In the diffuse parenchymatous form, there is a small-cell infiltration mostly leucocytic, and the muscle fibres appear pale and cloudy. In myocarditis, secondary to inflammation of the endo- or pericardium, bands of fibrous tissue tend to spread between the muscle fibres. Ulcerative endocarditis is apt to invade the myocardium locally, and in pyæmia small abscesses may form in the heart wall.

Clinical Features.—In the parenchymatous form præcordial distress with “fluttering” heart’s action, rapid and irregular pulse with a tendency to vomiting, restlessness, and even delirium are common. In all the other varieties the symptoms and signs are inseparable from those of the primary

condition, but feeble and irregular heart's action is certainly suggestive, and especially if cardiac dilatation be present. Fibroid myocarditis is essentially, in its chronic form, the same process as fibroid degeneration of the myocardium without much evidence of any pre-existing inflammation, and the condition is included under the heading of "Fibroid Degenerative Myocardium."

The **Diagnosis** must be guided by the advent of grave phenomena, suggesting a serious involvement of the heart muscle.

The **Prognosis** is always unfavourable, and death is generally due to cardiac failure.

The **Treatment** must be directed in part to the cause of the condition. Insist on absolute rest in bed, give stimulants cautiously, and apply counter-irritants (mustard blisters) over the præcordia.

PARENCHYMATOUS DEGENERATION OF THE MYOCARDIUM is a generalised condition, due to the toxins of certain organisms, in which the muscle fibres become pale and soft, and cardiac failure almost inevitably terminates life.

(2) FIBROID DEGENERATIVE MYOCARDIUM

This is due to anything interfering with the blood-supply to the heart muscle, or it follows where great hypertrophy of the heart wall has developed. It is similar in its result to the myocardial changes which follow adherent pericardium. Possibly alcohol and syphilis may cause fibroid changes in the myocardium.

The **Clinical Features** vary with the degree of involvement, but feeble and rapid cardiac action with pain is the most common. Little can be done by way of treatment. Tonics and potassium iodide are worth a trial.

(3) BROWN ATROPHY OF THE HEART AND OTHER DEGENERATIONS

Brown atrophy, so common in old age, waxy degeneration, calcareous degeneration, and the hyaline degeneration of Zenker, require no description here. Fatty degeneration is discussed later.

(4) FATTY HEART

(a) Fatty Infiltration of the Heart

A deposition of fat resulting from general obesity and associated, more generally, with adult or later life. It occurs on the surface, and also, in some instances, infiltrates the muscular substance separating the fibres which generally atrophy, and the fat may even reach to the endocardium.

The **Etiology** includes the many causes of obesity, and among these excessive drinking of malt liquors might be particularised.

Pathological Anatomy.—The fat is specially marked in those positions in which it is normally present in moderate amount, for example, along the line of the interventricular septum, the auriculo-ventricular groove, at the apex of the heart, and elsewhere.

Clinical Features.—There is evidence of enfeebled circulation, with breathlessness especially on climbing stairs. The first sound is often weak, and the pulse is poor in intra-vascular pressure. Patients with fatty heart are bad subjects for operation, and syncopal attacks occasionally supervene. More or less dilatation of the heart may also be present.

Diagnosis.—The general evidences of obesity should suffice.

Prognosis.—Fatty infiltration is amenable to treatment in cases not too far advanced.

Treatment.—Careful dieting, with little fatty, starchy, or saccharine foods. As a dry diet is most important, limit the amount of fluid taken per day to a minimum, and advise regular exercise with, in certain cases, Oertel's hill-climbing exercises. Give such tonics as iron (where required), arsenic, strychnine, and rarely digitalis.

(b) Fatty Degeneration of the Heart

A true fatty degeneration of the muscular fibres, most frequently affecting the left ventricle.

Etiology.—(1) Failing nutrition from old age; (2) wasting disease; (3) pernicious anæmia; (4) and certain poisons, and especially phosphorus, may cause a generalised fatty

degeneration. It is due also to local causes which act locally, such as atheroma of the coronary arteries, pericarditis, and myocarditis. It seems as if the heart muscle needed more oxygen than other muscular tissues.

Pathological Anatomy.—The muscular fibres are pale and soft, and show minute fat globules in rows, replacing the muscle substance; it is most marked in the left ventricle, where the typical thrush-breast-like appearance is well seen. The muscle becomes soft and friable, and hence the risk of rupture of the heart.

The **Clinical Features** include enfeeblement of the circulation, dyspnoea, often Cheyne-Stokes breathing, palpitation, and sometimes syncope. There is a tendency to yawning, sighing respirations, and giddiness; not infrequently a corresponding enfeeblement of the mental faculties and memory supervenes. A measure of dilatation of certain of the cardiac chambers may be present.

The **Diagnosis** can only be suspected by the nature of the case and the group of symptoms present.

The **Prognosis** is bad.

The **Treatment** must be on palliative lines. A quiet life should be ordered with the use of tonics such as strychnine and arsenic, and only with great caution is digitalis or strophanthus to be administered.

(5) MYOMALACIA CORDIS: ACUTE NECROSIS OF HEART

This is due to blocking of a branch or branches of a coronary artery, generally due to arterial degeneration. The coronaries are end-arteries, and therefore necrotic changes follow in the area of heart muscle deprived of blood. The muscle fibres lose their striæ and nuclei, and the whole patch affected becomes pale and yellowish in colour, and very friable. On the endocardium covering the infarction, blood-clot forms. It is most commonly met with near the apex of the left ventricle, and the weakened part may give way, leading to the formation of a cardiac aneurism with eventual rupture and death.

Under any circumstances acute softening is rapidly fatal.

X. CARDIAC ANEURISM

1. *Of a Valve.*—In ulcerative endocarditis this is not uncommon. The aneurism may perforate, and generally the aortic orifice is the one affected.

2. *Of the Heart Wall.*—This is rare. It may be due to a patch of acute necrosis (myomalacia cordis), or some other condition which weakens a part of the ventricular wall. It is most often met with near the apex of the left ventricle. The aneurism may grow to be nearly as large as the whole heart, and may even bulge the chest wall. It tends to rupture into the pericardial sac.

XI. RUPTURE OF THE HEART

THIS may result from fatty degeneration or infiltration, or from acute softening, or may be due to the rupture of a cardiac aneurism. It causes sudden death, with great distress and intense dyspnœa.

XII. CONGENITAL MALFORMATIONS OF HEART

AN arrest of development, which may sometimes cause conditions incompatible with life. Endocarditis may also be present.

There may be—

1. Deficiency or absence of septa, either the interauricular (patent foramen ovale) or interventricular (often at or near the undefended spot).

2. Alteration in number or arrangement of cusps. Where there are only two well-marked aortic cusps, the third cusp may be represented by a minute, undeveloped structure. Such a malformation is apt to predispose to endocarditis or degenerative changes at the affected orifice, which is generally either the aortic or pulmonary.

3. Persistence of foetal structures, of which the foramen ovale has already been mentioned; another is the ductus

arteriosus, which conveys the blood in the foetus from the pulmonary artery into the aortic arch. An imperfect division of the bulbus arteriosus is more rarely present. Sometimes stenosis of an orifice occurs, such as the pulmonary from mal-development.

4. Foetal endocarditis may cause lesions similar to those occurring in later life, only more frequently met with in the right heart. The type of endocarditis present is generally chronic.

The **Clinical Features** will depend on the gravity of the lesion present. In a case of a very widely patent foramen ovale, often associated with other congenital malformation, the cyanosis is very obvious, giving the typical *morbus cæruleus*. The extremities are blue and cold, the fingers and toes are clubbed at their extremities, and there may be a varying degree of dyspnœa. There is a tendency to great backward pressure in such a case, with venous engorgement or even pulsation in the veins of the neck, and often a pulsating liver. There is a predisposition to bronchitis, and if the blood-supply to the lung is very defective, to pulmonary tuberculosis. A patent ductus arteriosus is recognised by a loud, somewhat late systolic murmur which is heard most clearly just outside the pulmonary area, and which may be continued beyond the second sound. It is often accompanied by a thrill.

Where a murmur is present as the result of other congenital lesions, it is rarely accompanied by a thrill, but may be associated, either with no enlargement of the heart at all, or with marked enlargement of the right heart only.

The **Diagnosis** is easy in very obvious cases, which present the clinical features above mentioned and at an early age.

The **Prognosis** depends much on the lesion present. In a case in which there is great interference with the aeration of the blood, death is not uncommon from bronchitis, pulmonary tuberculosis, or mere inability to stand the wear and tear of life.

The **Treatment** should consist in the very careful upbringing of the patient, and the physician should order warm clothing, good food, and general tonics, especially cod-liver oil and iron.

XIII. DISEASES OF ARTERIES

(1) INFLAMMATIONS OF ARTERIES

(a) Acute Arteritis

MAY be (1) *Local*, where, for example, an artery is involved in an abscess; or (2) *General*, where, as in certain fevers, the arteries are extensively affected. The general form is met with during or after an infective fever, and especially diphtheria, typhoid, and influenza. Either the infective organisms or their toxins are believed to be the cause of the condition.

The **Pathological Anatomy** of the general form is the following:—Gelatinous patches of varying size appear in certain arteries. These consist of embryonic cells which are arranged in rows, and invade along the lines of the vasa vasorum.

Thrombosis may follow and even gangrene of the affected limb or limbs.

(b) Endarteritis Obliterans

A small-cell proliferation of the deeper layers of the intima. The lumen of the affected vessel may become absolutely blocked from thrombosis.

Etiology. — Syphilis certainly, and possibly alcoholism and malaria, are believed to be the commonest causes of the affection.

The **Clinical Features** vary. There will probably be pain, often intermittent, together with local cyanosis, cramps, and numbness of the extremities, while gangrene may eventually develop.

(c) Syphilitic Arteritis

Specially affects arteries at the base of the brain, and the preceding form of arterial disease (endarteritis obliterans) is the commonest syphilitic variety.

Pathological Anatomy.—Greyish-white, opaque nodules appear which are cartilaginous in appearance. They are due to a small-cell infiltration of the intima and the other coats,

and gummata may be associated. A syphilitic periarteritis sometimes occurs.

The **Clinical Features** are generally fairly definite, headache being common, and also paralysis, its extent depending on the part of the brain which is deprived of its blood-supply.

(d) *Tuberculous Arteritis*

Tuberculous arteritis is met with in pulmonary cavities or where tubercle nodules attack the wall of an artery, penetrating from without inwards, and eventually leading, in some cases, to diffusion of tubercle bacilli into the blood-stream.

(2) ARTERIO-SCLEROSIS (GULL and SUTTON)

A diffuse or localised thickening of the intima of the arteries, secondary to primary changes in the media and adventitia. The term arterio-sclerosis is very variously employed, and atheroma is often closely associated with it, and has been described as one of the forms of the disease. Atheroma will, however, be described separately, and the term arterio-sclerosis is here used in a limited sense.

Etiology.—It may be the result, *firstly*, of senile changes in vessels, often occurring at an early age in certain families. It should be remembered that in this group of cases atheroma is an almost necessary part of the process, and is really inseparable from it. *Secondly*, it may follow long-standing abuse of alcohol, and also arise from the effects of lead, gout, and syphilis. *Thirdly*, overstrain, and almost certainly over-eating, predispose to its development; and, *fourthly*, in chronic Bright's disease, arterio-sclerosis is an important sequel.

Pathological Anatomy.—There may be thickening of all the coats of the artery, and pathologists differ with regard to the coats that are first affected. The thickening of the intima referred to in the opening sentence, is a small-cell infiltration which represents nature's effort at strengthening an artery wall, whose other coats (media and adventitia) have been weakened. This thickening may be limited, or may involve a considerable extent of the vessels. Where the disease is wide-spread, there is invariably associated hypertrophy of the left ventricle and a cirrhotic change in the kidneys; and primary cirrhosis of the

kidney is in reality a part of the disease, in which the arterio-sclerosis in the vessels and the enlargement of the left ventricle are also leading features, although it is probable that the condition may be primary in the vessels.

A diffuse form of arterio-sclerosis is often seen in middle life, the change, mainly a hyaline one, beginning in the media and involving the muscular fibres and the elastic tissue; the intima over the affected part undergoes thickening. A similar sclerosis may involve the veins in these cases.

Unquestionably a small-cell infiltration is the initial lesion, and it is thought that it passes along the lines of the vasa vasorum.

Clinical Features.—There is marked thickening, with localised dilatations and constrictions of many of the arteries, and it may be easily recognised on palpating the radial artery in a typical case. The pulse is one of high tension, and the enlargement of the left ventricle is almost constant. The aortic second sound is loudly accentuated, and the mitral first is generally of a booming character. The thickening of the vessels may, however, be in part due to what has been called “hypertonus,” by which is meant vasomotor constriction. This temporary increase of intravascular pressure, which results from the presence of ptomaines circulating in the blood, is a common feature in cases of marked constipation, even where the vessels are absolutely normal, but hypertonicity adds to the pressure in the vessels in arterio-sclerosis, in which condition toxins are apt to be present in the blood-stream. The patient suffers from headache, and often from sleeplessness, and there is always a risk of the combination of high tension with weakened vascular walls leading to cerebral hæmorrhage. Sometimes thrombosis occurs, where the degenerative changes in the cerebral arteries are very pronounced. The urine is generally excessive, and closely corresponds, in amount and character, with the urine excreted in primary cirrhosis of the kidney.

The **Diagnosis** is rarely difficult, the examination of vessels and heart generally aiding in the recognition of a well-marked case.

The **Prognosis** largely depends on the possibility of counteracting the etiological factors responsible for the onset of the disease.

Treatment.—Purgatives should be freely administered, overstrain should be prohibited, and alcoholic tendencies kept in check. The possibility of syphilis and of lead poisoning should not be forgotten. Careful dieting is important, and a remedy often of the greatest value is iodide of potash, while the nitrite group, as, for example, nitroglycerine in doses of $\frac{1}{100}$ th of a grain, often relieves the patient's symptoms.

ATHEROMA

Atheroma cannot be entirely separated from the preceding condition of arterio-sclerosis. It is essentially a senile change, but many diseases, such as gout, alcoholism, and syphilis, and constant overstrain tend to produce or increase the disease.

Pathological Anatomy.—Slight degrees of atheroma are present in the arteries of all persons after, or even before, middle life. There is a thickening of the inner part of the intima, in which fatty and, eventually, calcareous deposition occurs, and lime salts may be deposited in very large quantities, the disease interfering seriously with the lumen of the vessel. Sometimes atheromatous cysts or abscesses occur in connection with these patches, and the patches are best marked at the commencement of the aorta and in the larger arterial trunks. The middle coat of the affected vessels becomes weakened, so that an aneurism is not infrequent, and sometimes a dissecting aneurism is caused by the blood passing behind a calcareous plate and penetrating between the coats of the vessel. In the smaller arteries, atheroma is often associated with arterio-sclerosis, and there is a tendency to thrombosis occurring, which, in the vessels of the brain may cause extensive softening, and in the vessels of the extremities may produce gangrene.

The **Clinical Features** vary greatly. The tortuous, thickened radial arteries with calcareous plates in the walls, can often be well palpated with the finger, and the loss of expansile power, in a marked case, is very typical. There is generally a systolic aortic murmur, frequently due to atheroma of the aortic cusps. The interference with the blood-supply to the organs of the body, and also to the extremities, gives rise to symptoms and signs varying in different cases, and varying with the degree of the interference. Many old patients complain of coldness of

the extremities, and gangrene as already mentioned is not infrequently a final phenomenon.

The **Diagnosis** is generally easy, and the **Prognosis** depends on the degree of the condition.

The **Treatment** is unfortunately *nil*. All that can be done is to preserve life, and to treat symptoms as they occur.

(3) ANEURISM

There are various types of aneurism which require a few words of description.

1. A *True* aneurism is one in which the sac is formed by the coats of the vessel.

2. A *False* aneurism has its sac formed by tissues outside the artery, the walls of the vessel having been eroded.

3. A *Dissecting* aneurism is one in which the blood finds its way between the coats of the artery, and either passes back again into the vessel or ruptures externally.

4. An *Arterio-venous* aneurism is an aneurism which communicates with a vein.

In shape, aneurisms may be *Fusiform* or *Saccular*; in the former the vessel coats give way all round, in the latter the bulging is at one weak spot, and does not necessarily involve the whole circumference of the vessel.

Etiology.—Aneurisms may result from two groups of causes: (1) Local weakness of the arterial wall, and (2) Increased blood pressure.

(1) Local weakness may be due to arterio-sclerosis, and especially before compensatory thickening occurs. The dilatation may be a general yielding of a larger or smaller stretch of vessel, or may be quite localised. Anything inducing arterial degeneration may cause weakening, and injuries are sometimes responsible; occasionally pyæmic emboli produce local weakening of the vessel wall and dilatation. (2) Increased blood pressure is usually the result of overstrain, and cirrhotic changes in the kidney may be an important factor in the case.

(a) Intrathoracic Aneurisms

Etiology.—They occur mostly in men between 35 and 50 years of age, and are rare before 20 and after 50.

1. *Aneurisms of the ascending part of the Aortic Arch.*—These may occur close to the orifice, and generally in one of the sinuses of Valsalva, and as the result of ulcerative endocarditis. They are of small size, and may give rise to no symptoms until they rupture into the pericardial sac, pulmonary artery, or elsewhere.

If the aneurism is higher up, it is larger, and may be either a fusiform dilatation, or more rarely saccular. It may erode ribs or sternum, and may bulge in the 1st, 2nd, or 3rd intercostal spaces on the right side.

Physical Signs.—On *Inspection* a pulsating, expansile tumour may be seen. On *Palpation* an impulse is sometimes palpable, and is expansile, while a thrill may be felt. *Percussion* generally yields a definite increase of dulness to the right of the sternum in the 2nd and 3rd spaces. On *Auscultation* a systolic murmur may be present over the sac. There may in some cases be aortic incompetence, with an aortic regurgitant murmur. The left ventricle is enlarged if the aortic orifice is interfered with, but is generally unaffected as regards size, although the heart may be pushed downwards by the tumour. Rarely the aneurism presses on the superior vena cava, causing distension of the veins in the neck and perhaps also in the chest.

The *Respiratory System* is as a rule unaffected; sometimes the right bronchus is pressed upon, but this is rare.

In the *Integumentary System* there are localised changes in the skin covering the aneurism, should there be very marked external bulging. The skin becomes red and thin, and may be covered by blebs which burst and sometimes lead to ulceration. Rarely an aneurism bursts externally, although it sometimes does so, and there may be leaking of blood or weeping, as it is called, prior to the final rupture.

Pain is usually only a marked feature when there is pressure on bone or nerves.

2. *Aneurism of the transverse part of the Aortic Arch.*—This aneurism often involves the innominate artery. Sometimes these aneurisms are saccular and bulge into the neck, sometimes they are very large, and may erode the manubrium sterni, and may form a large tumour with several accessory sacs.

Physical Signs.—On *Inspection* we note the pulsating expansile tumour in the neck or in the region of the **manubrium**.

Palpation demonstrates the pulsation and sometimes also a thrill. *Percussion* generally yields a very definite area of dullness, and *Auscultation* may reveal a systolic bruit. The pulse is often very typical, should the right subclavian be weaker than the left. This may be the result of the innominate artery being interfered with in, or near, the sac in one of the following ways:—(a) Its orifice may be blocked with clot, (b) the aneurism may compress the innominate or subclavian distally, or (c) by the large size of the sac there may be great loss of force as compared with the left subclavian, should it not be also involved in the sac. A sphygmographic trace of the two pulses is often distinctive and helpful.

In the *Respiratory System* there is frequently pressure on the trachea or left bronchus, and the aneurism may weep, and eventually rupture, into the bronchus. Dyspnœa is common, either from this tracheal compression, or from pressure on the vagus, the pulmonary plexus, or the left recurrent laryngeal nerve. The stridor and typical brassy cough are generally very marked, and sometimes the dyspnœa becomes paroxysmal. The brassy cough is due to irritation of the recurrent laryngeal, or, according to Semon, to contracture following paralysis. Should a low type of pneumonia ensue, purulent foci develop in the damaged areas of lung, while bronchiectasis may result. Tracheal tugging is a valuable physical sign. The patient is asked to hold back the head, and the cricoid is grasped by the fingers and pushed upwards; with each contraction of the ventricle the aneurism drags on the trachea, if it is adherent to it or to the left bronchus, which passes under the aortic arch.

In the *Alimentary System* pressure on the œsophagus may lead to dysphagia.

The *Nervous System* is specially important. Pressure on the vagus and recurrent laryngeal nerves has already been referred to. The sympathetic may also be compressed. It supplies the dilator pupillæ, and therefore, if the nerve is irritated, dilatation of the pupil will occur on the same side; if paralysis, then contraction of the pupil ensues. The left phrenic nerve is occasionally involved, leading to paralysis of the left half of the diaphragm.

Pain must be a prominent symptom, due to pressure on some of these important structures mentioned above. It may **shoot down the left arm** and have a definite anginous character.

3. *Aneurism of the descending part of the Arch.*—Here the aorta is deep-seated and approaches the spinal column. It is therefore less likely that any evidence of the aneurism will be present on physical examination, unless the vertebral bodies are eroded (4-6 Dorsal), and unless there are definite signs of the tumour in the back; the latter are often elicited by percussion.

This is an aneurism of symptoms, and pressure symptoms will be more or less marked. The left bronchus, the œsophagus, and possibly some of the nerves mentioned above, may be affected, and in any case pain is very commonly present. Probably the eroding of the vertebral bodies is responsible for a considerable part of this, and in a few cases, including one which the author saw post mortem, the blood makes its way into the spinal canal, causing complete paralysis of all four limbs.

The table appended gives a good idea of how aneurism cases terminate. Experience, however, teaches that they all kill by pressure more frequently than by rupturing internally or externally, and probably many aneurisms which are never diagnosed prove fatal in this way.

TERMINATION OF ANEURISMS OF AORTIC ARCH¹

CRISP'S TABLE

This table refers to rupture of aortic aneurisms into structures named, unless where otherwise stated.

| 98 Cases. | | 48 Cases. | | 21 Cases. | |
|--------------------------|----|-------------------------|----|-------------------------|---|
| <i>Ascending Part.</i> | | <i>Transverse Part.</i> | | <i>Descending Part.</i> | |
| Pericardium . . . | 30 | Trachea . . . | 4 | L. Pleura . . . | 4 |
| Externally . . . | 6 | Pericardium . . . | 2 | R. Pleura . . . | 4 |
| Left Pleura . . . | 4 | R. or L. Pleura . . . | 4 | œsophagus . . . | 5 |
| Fatal Pressure on | | Bronchus . . . | 2 | L. Bronchi . . . | 2 |
| Trachea and Bronchi | 7 | œsophagus . . . | 2 | Pressure on Trachea | |
| Hydropericardium and | | Pressed fatally on | | and Bronchi . . . | 2 |
| Hydrothorax . . . | 6 | Trachea and | | | |
| œsophagus . . . | 2 | Bronchi . . . | 12 | | |
| Vessels { Pul. Art. . . | 3 | Suffocation . . . | 3 | | |
| { Sup. Cava . . . | 3 | Hydrothorax . . . | 1 | | |
| Ventricles { Right . . . | 2 | | | | |
| { Left . . . | 1 | | | | |
| Trachea . . . | 1 | | | | |

Diagnosis.—Definite physical signs are an enormous help. In a *solid tumour* the pressure is constant, unvarying in degree,

¹ Table quoted from the late Sir T. Grainger Stewart's *Lectures on Medicine*.

and the pulsation is not expansile. In an aneurism the pressure varies; it is more like a living, constantly changing pressure, and the tumour, if palpable, is expansile.

Age, sex, and kind of work may help in the diagnosis, and an X-ray examination is of enormous advantage.

An *encysted pleural effusion* can hardly be mistaken.

Prognosis.—An aneurism can be cured. It becomes hard, and does not pulsate so vigorously, which means that clotting has occurred. But there is always a danger that fresh sacs may develop. In an advanced case, never place the duration of life at more than a few months. It must not be forgotten, however, that a fusiform dilatation of the aorta is compatible with a long and useful life.

Treatment.—Try to aid clotting in the sac, which may be done—

1. By rest, keeping the patient in bed for weeks or months, and even insisting on the prone position being constantly maintained during that time.

2. By low diet and a minimum of fluid. Tufnell's method of treatment consists in 10 oz. of solids and 8 oz. of total liquids per day. Tufnell combined this starvation diet with rest in bed for 2 to 3 months.

3. By administering potassium iodide in doses of from 10-90 grains thrice daily. Iodism is often prevented by doubling the dose the moment it threatens to appear, but with certain patients a considerable interval is necessary before the drug can be again taken.

4. By various surgical measures which have been tried to induce clotting. These include—the introduction into the sac of fine iron or gold wire, or horse-hair; scratching the inner wall of the sac with a fine needle; electrolysis, either the positive pole alone, or both poles being introduced into the sac of the aneurism.

Recently gelatine has been used hypodermically or per rectum. A 100 c.c. of a 2 per cent solution is administered, and if given by the skin the solution must be very carefully sterilised. At least 15-20 injections are necessary.

Bleeding has been employed where there is much pain and pulsation in the aneurism (taking 10-15 oz. of blood from the arm), but the method is not very frequently resorted to.

Under treatment by rest, low diet, and potassium iodide, very great improvement results, pain disappears or is much relieved, pulsation lessens, and the aneurism tends to become harder. A few surgical authorities commend the wiring of the sac, the gelatine method having been found disappointing. An innominate aneurism may be treated by ligaturing the subclavian and common carotid, an operation which has proved successful in some cases.

A few *special symptoms* must be referred to.

Pain, if very severe, demands the use of opium; *thirst*, when the dry diet is in force, is relieved by sipping a solution of citric or other suitable acid in water; and *sleeplessness* may call for the administration of bromides or other hypnotic.

Where an aneurism is bulging through the skin, elastic pressure on the tumour should be tried, while painting on collodion over cotton wool may help to support the sac.

(b) Abdominal Aneurism

It is most commonly situated at the coeliac axis branch just below the diaphragm—more rarely lower down at, or involving, the superior mesenteric or renal arteries. The aneurism may be saccular or fusiform. Pain is common, either dull and aching, or sharp and lancinating, and is often due to involvement of the spinal nerves. The aneurism may erode the vertebral bodies, and sometimes it grows to a large size.

The *Physical Signs* include very definite pulsation, which is expansile; there may be a thrill, and often a systolic murmur. These physical signs are not easily made out in aneurism of, or at, the coeliac axis artery. Sometimes it is possible, by comparing the pulse in the femorals with that in the radial arteries, to recognise a distinct retardation and diminished tension in the former as compared with the latter.

The expansile character of the pulsation in the aneurism serves to distinguish it from a solid tumour.

The aneurisms may rupture into the peritoneum, and sometimes into the stomach, and are seldom cured.

Treatment.—Combine absolute and prolonged rest with a low diet and potassium iodide. If the aneurism is low enough in position, proximal pressure might be tried; but it is

severe treatment, and frequently causes shock, and as the pressure must be kept up for hours, gangrene of the legs may result, while the pain is usually excruciating. Opium is generally requisite to soothe the patient during the application.

XIV. DISEASES OF THE VEINS

VARICOSE veins belong to the department of surgery, but it is necessary to refer briefly to phlebitis.

Phlebitis, or inflammation of the walls of a vein, may be of primary origin, and is believed to be more common in rheumatic or gouty patients. It is not infrequent in anæmia, and may be a very undesirable complication in one of the continued fevers. Very commonly it is secondary to pressure on the veins in the pelvis, or pressure on the vein above the level of the phlebitis, but an injury to the vein, such as a blow, is an important exciting factor in many cases, and such injuries are more apt to occur in connection with varicose veins. The vein wall is painful, and generally thrombosis has occurred or is in process of occurring. The veins most frequently affected are the femoral or popliteal. The risk of a portion of clot separating from the thrombus and finding its way to the lung should ever be kept before the mind.

The **Prognosis** is favourable in most cases, unless the thrombus becomes purulent, when it forms an abscess in a most undesirable position.

Treatment.—Absolute rest is imperative, with the application of sedative lotions such as lead and opium. The limb should be supported, and if possible the affected part kept at a higher level than the patient's head. Bandages are of value, but in many cases the removal of the thrombosed and varicose veins is the wisest procedure. Should an abscess form, it must be opened in the usual way.

DISEASES OF THE RESPIRATORY SYSTEM

I. DISEASES OF THE NOSE

(1) CORYZA ; ACUTE NASAL CATARRH

Etiology.—There is a hereditary predisposition in some families, while lifelong coddling usually results in numerous attacks of nasal catarrh. The exciting causes are exposure to cold, wet feet, irritating vapours, and, in many cases, infection from other sufferers. *specific miasm*

Pathological Anatomy.—There are hyperæmia and swelling of mucous membranes, with changes in the secretory glands, causing them first to cease secreting, and later to secrete a copious amount of clear mucus, which later still becomes muco-purulent. As a sequel, there may be a hypersensitive condition of the mucosa, with increased tendency to fresh attacks, and the catarrh may spread to the pharynx and gastro-intestinal tract, or to the larynx and bronchi.

Clinical Features.—A cold generally begins with sneezing, then dryness, loss of sense of smell, uneasiness and irritation in the nostrils, followed by a copious mucous, and eventually a muco-purulent secretion. The catarrh may spread to the pharynx, larynx, and conjunctivæ. It may affect the frontal sinuses and Eustachian tubes, causing headache in the former case, and deafness in the latter, with possibly either suppuration from infection, or indrawing, and even rupture, of the tympanic membrane from exhaustion of the air in the middle ear. The catarrh may spread to the bronchi (bronchitis) or to the alimentary tract. There is often some slight pyrexia and nearly always more or less general discomfort. Many patients have an associated herpes labialis coexistent with the attack of coryza.

Sung

The **Diagnosis** presents no difficulty. Occasionally a continued fever such as measles begins with coryza, while in cases really of *influenzal* origin, the aching bones and the exhaustion are typical of that disease.

Prognosis.—In acute cases, good; in chronic cases, or where there is hereditary predisposition, the condition may be obstinate.

The **Treatment** is frequently carried out without recourse to medical advice. In some cases, try to abort the attack by a hot bath or foot-bath, followed by a hot drink and, just before sleep, a dose of compound ipecacuanha powder (gr. 5-10). A saline purge in the morning completes the attempt at cure. Diaphoretics and diuretics are helpful, and locally adrenalin (1 part in 1000 of $\frac{1}{2}$ per cent chlorotone), introduced into the nostrils on pledgets of wadding and sniffed up the nose, is of great value, while cocaine 5-10 per cent is sometimes sprayed or painted on. Inhaling carbolic acid, eucalyptus, or menthol is commended. Many people take tincture of camphor in drop doses on sugar.

Adrenalin solution 1:1000

(2) CHRONIC NASAL CATARRH

This is simply a chronic form of the preceding, and it may require long and persistent local and general treatment to effect a cure. Often there is a hypertrophic swelling of the turbinated bones, tending to block the Eustachian tubes and so cause deafness, and, not infrequently, adenoids are associated, the patient becoming a mouth breather, and snoring dreadfully when asleep. A second variety is the atrophic, which is often associated with a most offensive discharge (*ozena*), and may result from caries, syphilis, the presence of a foreign body, etc.

The **Treatment** consists in relieving the hyperemia of the hypertrophic form either by the electric cautery, or by applying astringents (alum or silver nitrate solutions). In *ozena* the nasal douche must be resorted to, and boracic acid, listerine, or other antiseptic lotion used regularly.

(3) DISEASES OF THE SINUSES ASSOCIATED WITH THE NOSE

The antrum frequently becomes inflamed as the result of a decaying tooth, the root of which may penetrate into its

sinus
in
antrum
perforation
of sinus
cavity. An abscess may thus form, readily recognised by the pain and discomfort produced, and also by the darkness of the affected side when a suitable electric lamp is introduced into the mouth, the patient being examined in a dark room. In such a case the empyema or abscess of the antrum demands treatment by drainage, and for this purpose the offending tooth should be extracted and a tube inserted connecting the antrum with the mouth; by this tube the cavity may be washed out and thoroughly drained.

The frontal sinuses are sometimes affected as the result of an ordinary coryza, and this probably depends on the ease with which the infective organisms can pass upwards through the channel of communication with the nose. It is rarely necessary to resort to any special treatment other than that recommended for the relief of coryza.

(4, EPISTAXIS, OR BLEEDING AT THE NOSE

epistaxis
due to
systemic
disorder
This is a symptom of many morbid conditions. It may be the result of some local lesion such as occurs from trauma, polyp, local ulceration, or a varicose condition of the smaller vessels in the nostrils; or it may be one of the clinical features in a blood disease such as pernicious anaemia, chlorosis, leucocythæmia, or hæmophilia. It is also a common feature in the early stages of certain of the continued fevers in children, and especially scarlet fever, measles, and typhoid. Vicarious menstruation sometimes causes epistaxis, and it is unquestionably a frequent phenomenon in cases of marked backward pressure, whether from heart or lung disease. In certain families there is a tendency to nose-bleeding during adolescence, any slight strain apparently inducing a severe hæmorrhage. Towards old age, or where the vessels are markedly degenerated, there may also be alarming and frequent attacks of epistaxis. In a rarefied atmosphere epistaxis is common, and it adds much to the difficulties attending the ascent of very high mountains.

It is sufficient as a general rule to make the patient lie down on the floor or on a couch, to extend the arms above the head, and to apply something cold to the back of the neck. An ice-bag certainly acts most effectually. It is sometimes necessary to prevent the blood from flowing down the throat by turning the head towards the bleeding side so as to permit

of the escape of the blood from the nostrils. Plugging the nostrils with cotton wool soaked in a solution of suprarenal gland, such as adrenalin, and also, where necessary, plugging the posterior nares, causes speedy arrest of the hæmorrhage. In milder cases, pressure applied to the bridge of the nose or application of cold to the same region, may be sufficient. Remember that after plugging the nares blood is apt to undergo decomposition changes, and that it is wise therefore to wash out the nasal cavity with an antiseptic solution, and sometimes to use for the purpose of plugging, iodoform or other antiseptic gauze in preference to simple wool. In many cases, the condition of the patient's blood requires vigorous and systemic treatment, by means of iron, arsenic, or other remedies.

(5) NASAL POLYPI

These may be mucous or fibrous; the former are readily removed by means of the wire snare or even a pair of forceps. Fibrous polypi and malignant growths belong to the realm of surgery.

(6) HAY FEVER

Etiology.—There are three factors, all of which may be present in a case of hay fever. These are: (1) A neurotic constitution; (2) A nasal abnormality consisting in a hypertrophic rhinitis, or at the least a great hypersensitiveness of the nasal mucosa; and (3) Hay pollen, to a less extent the pollen of flowers, or, in persons with a special idiosyncrasy, the dust from the horse, cat, or other animal.

There is a powerful poison in the pollen, and especially in that of certain grasses. This has been isolated by Dunbar, and an antitoxin obtained from animals to whom the poison has been administered hypodermically.

Pathological Anatomy.—There is sometimes hypertrophic rhinitis.

Clinical Features.—The attack, usually associated with the season when hay seed is ripe, begins with a severe coryza, generally with tremendous fits of sneezing, the eyes stream, and the conjunctivæ may become inflamed. Sometimes the frontal sinuses are affected and severe headache results, sometimes the bronchi participate and bronchitic asthma ensues.

and indeed forms a distinct variety of hay fever. The condition lasts throughout the hay season; once established, it is hard to get rid of it, and it is apt to recur annually.

The **Diagnosis** requires no remark. *Easy.*

The **Prognosis** depends on the possibility of avoiding the cause, and on the success attending the attempt to cure the hypersensitiveness and to strengthen the patient's general health. *Good*

Treatment.—Send the patient to the sea-side, or, best of all, on a sea voyage during the hay season; sometimes a high altitude is beneficial. For the attack, cocaine in 10-20 per cent solution, or adrenalin, should be painted on, or introduced on pledgets of cotton wool into, the nostrils. An attempt should be made to lessen the hypersensitiveness by the use of astringents, or by the galvano-cautery, by which many small vessels are obliterated, thus reducing the hyperæmia. Dunbar's antitoxin is beneficial in some cases, and is applied locally to the affected mucous membranes. Lastly, attend to the patient's general health, administering such tonics as arsenic, phosphorus, and strychnine.

II. DISEASES OF THE LARYNX

(1) ACUTE LARYNGITIS

AN acute catarrhal inflammation of the larynx.

Etiology. — Exposure to cold, and especially a sudden change from overheated to cold air, excessive use of the voice, irritating vapours, entrance of irritating substances and fluids, and propagation of neighbouring inflammation (*e.g.* secondary to severe coryza or bronchial catarrh).

There is a hereditary predisposition to coryza, pharyngitis, and laryngitis; the coddling of children is apt to increase any such susceptibility.

Pathological Anatomy.—There is reddening of the larynx, specially of the posterior ends of the true cords, false cords, and inter-arytenoid fold, with increased secretion of mucus or muco-pus. Sometimes superficial ulcers form. It may go on to œdema under certain conditions.

Clinical Features.—These include dryness of throat, hoarseness, tickling cough, which is later often spasmodic, and

œdema.

occasionally pain, but only, as a rule, on swallowing, when the epiglottis may cause the discomfort. There is generally ~~no~~ fever. In children, inspiration is apt to be croupy, and laryngismus stridulus may develop.

Diagnosis.—Little difficulty should be experienced in diagnosing this condition from *laryngeal diphtheria*. The recognition of the organism and the presence of albumin in the urine ought to point to diphtheria.

Prognosis.—Generally recovery occurs, but the condition may become chronic; if œdema ensues, it may cause death by asphyxia.

Treatment.—Absolute rest to the voice, and keep the patient in one atmosphere, and, if necessary, in bed. A diuretic and diaphoretic mixture may be given with a purge in the morning. Warm drinks, such sedatives as antimonial and ipecacuanha wine, are beneficial for the cough, and, above all, the inhalation of steam, with or without some agent added. Compound tincture of benzoin, the succus conii, and creosote are excellent for this purpose. Ammonium chloride is often inhaled with benefit. The wet pack is an effective remedy when used at bedtime. It consists of a piece of wet lint or a wet handkerchief wound round the throat, and covered with a piece of indiarubber tissue or oiled silk, and on the top a flannel bandage.

Cocaine (10 per cent solution), painted on, may abort the attack, and is worth a trial.

Ammonium chloride in *diphtheria* *Septicæmia*
 30 - 35 (2) ŒDEMA OF THE GLOTTIS

There are two kinds of œdema of the glottis — (A) inflammatory, and (B) non-inflammatory or dropsical.

A. Inflammatory Œdema is the result of "septic" laryngitis, often of erysipelatos nature. The epiglottis, ary-epiglottic folds, and false cords become enormously swollen, and the œdema may be, although rarely, subglottic in position.

The **Clinical Features** are alarming dyspncea, threatening complete asphyxia, in addition to the phenomena of acute laryngitis, and naturally pain is often a very pronounced symptom.

The **Diagnosis** can readily be made with the finger, by which the swollen œdematous epiglottis may be easily palpated.

Acute Hemorrhagic Edema has marked the larynx.
Edema due to K1. Frequent Sp. time.

The **Prognosis** should be guarded, and must depend on success of treatment.

The **Treatment** should be prompt and energetic. Scarify the epiglottis with a curved, sharp-pointed bistoury, the blade of which should be guarded with adhesive plaster. if this fails to afford relief, tracheotomy or intubation must be resorted to. During the early stages try to arrest the œdema by the use of ice to suck and the inhalation of moist air, and give iron and quinine internally.

(Edema may sometimes follow in tubercular and syphilitic laryngitis giving rise to similar symptoms, and in such cases it calls for the same treatment.

B. Non-Inflammatory Edema may occasionally occur in Bright's disease, sometimes in heart disease, and more rarely from other causes. It may require surgical treatment.

(3) CHRONIC LARYNGITIS

A chronic catarrhal inflammation of the larynx.

Etiology. — It may result from an acute attack, but is more often due to excessive use of the voice (hawkers, clergymen, and singers). It may result from cold, and with the pharynx from alcohol or tobacco in excess, and especially from cigarette smoking.

Pathological Anatomy. — Swelling and thickening of mucous and submucous coats of the true and false cords, the inter-arytenoid fold, and the epiglottis, frequently with superficial ulceration and a granular appearance of the true cords.

Clinical Features. — Similar to the acute form, but more hoarseness, dryness, and cough; it is often associated with pharyngitis.

Diagnosis. — Care should be taken to exclude tubercular disease of the larynx.

Prognosis. — Recovery under favourable conditions. it is sometimes permanent; œdema rarely follows.

The **Treatment** includes absolute rest for the voice, with a change of air, and especially departure from cold, damp fogs to the clear air of, for example, the Riviera, Egypt, or elsewhere. Sometimes the sea-side suits, and sometimes a high altitude is preferable. Avoid any irritant which may help to keep up

the condition, and especially spirituous liquors, highly spiced foods, and tobacco. Occasionally it is found that our patients are mouth-breathers, some nasal affection preventing the air from entering and being duly heated in its passage through the nostrils. In this case attention should be directed to the nose. Various pigments may be applied to the larynx, such as: tannic acid in 4-10 grains to the ounce solution, zinc sulphate 1-5 grains to the ounce, and in very obstinate cases, silver nitrate 10-50 grains to the ounce of distilled water. Steam impregnated with oil of pine, turpentine, eucalyptus, and similar remedies may be inhaled with benefit.

(4) LARYNGISMUS STRIDULUS; SPASMODIC CROUP;
CHILD-CROWING

Spasmodic constriction of the adductors of the glottis, often with carpo-pedal spasms.

Etiology. Common in rachitic and weakly children, but is directly excited by irritation of the stomach, by dentition, and similar causes.

Clinical Features.—May commence with crowing inspiration, generally nocturnal, followed by cessation of breathing, early pallor and later cyanosis of face, and bending back of the head. The spasm yields with a loud crowing inspiration. There may be carpo-pedal contractions, while occasionally death results before free respiration is restored.

Prognosis.—Generally favourable.

Treatment.—(1) For the relief of an attack nothing is so helpful as a hot bath, or even a sponge wrung out of hot water placed over the upper part of the chest. In other cases cold water may be dashed on the face and chest, and smelling salts held to the nose. Chloroform anaesthesia has been employed in very severe cases. Where all these measures fail, tracheotomy may be performed. (2) Remove the cause of the attack, whether it be stomach disorder or dental irritation. (3) Give constitutional remedies such as cod-liver oil, syrup of the iodide of iron, and so forth.

(5) PHTHISIS LARYNGEA

In pulmonary tuberculosis from 13 to 97 per cent of patients have an affection of the larynx. The discrepancy

according to Henige

25

25

25

between these figures is due to the fact that in the great majority of cases the laryngeal changes are of a trivial nature, and the authorities who accept the smaller percentage do not take such cases into consideration. Anæmia of the larynx and a catarrhal laryngitis, somewhat resistant to treatment, are common in pulmonary phthisis, and they make up the difference. The tubercular affections to which the term phthisis laryngea properly belongs, are ulceration and infiltration, and sometimes a resultant perichondritis. Very rarely is the tubercular involvement primary; it is generally secondary to infection of the lungs. Laryngeal phthisis is most common between the ages of 20 and 40, and is rare in children.

Pathological Anatomy.—Tubercles appear in mucous and submucous tissues, with surrounding inflammation, and they lead to ulceration. The sites of the disease are the inter-arytenoid fold, the false and true cords, the arytenoids, the ary-epiglottic folds, and the laryngeal surface of the epiglottis.

The **Clinical Features** closely resemble chronic laryngitis, with hoarseness of voice and short cough, but the special symptom is pain on swallowing. Aphonia may result from temporary paralysis, from fixation of an arytenoid or a cord, and, in advanced cases, from destructive ulceration. Where there is much œdema of the glottis, the dyspnœa is extreme.

The **Diagnosis** is usually fairly easy.

*Tubercular Affections of
the Larynx.*

*Syphilitic Affections of
the Larynx.*

- | | |
|--|--|
| 1. <u>Pain.</u> | Little pain. |
| 2. Greyish-white colour, especially of ulcers. | Bright red. |
| 3. Breaks down later. | Breaks down earlier. |
| 4. Laryngeal surface of the epiglottis affected. | Edge and lingual surface of the epiglottis affected. |

The **Prognosis** is unfavourable, and the duration of life is often limited to 6 months; sometimes it extends to 3 years. The short duration of life is due to the fact that the lungs are often seriously involved before the larynx suffers, and also that tubercular ulceration heals badly, and relapses are apt to occur.

Treatment.—Pain on swallowing may be relieved by

giving ice to suck, before administering food, while liquids and semisolids are more easily swallowed than solids. In very severe cases, a 10 per cent solution of cocaine should be painted on, or a quarter of a grain of morphia insufflated in the form of a powder mixed with starch.

Try to secure healing of the ulcers. Some authorities recommend lactic acid applied in a strength of 20 to 80 per cent, others prefer the cautery, but both methods are unsatisfactory. Antiseptics, such as carbolic acid, iodoform, guaiacol, or menthol in olive oil, and similar remedies, may be applied locally.

In cases which are not too far advanced, pure, dry air affords the greatest comfort—either the high altitudes of the Swiss mountains, or the warm, sunny winter climate of Egypt or Algiers. Caution the patient about over-use of the voice, and recommend abstinence from stimulants and irritants so far as possible.

(6) SYPHILITIC DISEASE OF THE LARYNX

Generally a later manifestation of syphilis, and the following conditions may be present:—

1. **CONDYLOMATA.**—Flat, smooth, yellow patches, situated on the true cords, epiglottis, false cords, and inter-arytenoid folds. Not very frequent in occurrence.

2. **GUMMATA.**—On ary-epiglottidean folds, false and true cords, epiglottis, rarely subglottic in position.

3. **ULCERATION.**—Comparatively superficial, if secondary to condylomata; deep, if secondary to gummata.

4. **PERICHONDritis.**—Secondary to any of the previous conditions.

Clinical Features. — Hoarseness, aphonia, sometimes dysphagia, much less pain than in tubercular disease. Cicatrisation frequently follows, and may cause great trouble by interference with the glottis.

Diagnosis.—See Phthisis Laryngea.

The **Prognosis** is good if treated, but tertiary syphilis not infrequently causes the formation of membranes and stenosis.

Treatment.—As serious syphilitic affections of the larynx are really of tertiary nature, iodide of potash is the best remedy, although mercury may also be administered. Where cicatriza-

tion occurs, it may be found necessary to dilate with a laryngeal bougie, or even to perform tracheotomy.

III. DISEASES OF THE BRONCHIAL TUBES

(1) ACUTE BRONCHITIS

AN acute catarrhal inflammation of the mucous membrane of the bronchial tubes, which may involve either the large or the small tubes; in the latter case the condition is called capillary bronchitis.

Etiology.—One *predisposing* cause is delicacy, especially in the young or the old, and perhaps chiefly in young persons the subjects of rickets. Gout, Bright's disease, heart disease, and many of the continued fevers may also be termed predisposing conditions, although in such ^{whooping cough & measles} fevers as measles and whooping-cough a bronchitis, due to the local action of the specific organism, is a leading clinical feature of the fever, which might therefore be termed an exciting cause. *Staphylococci*

The *exciting* causes are cold and damp, irritating vapours, foreign bodies, and inflammations of the larynx and nose. Bronchitis frequently results from coryza, and is by no means an uncommon part of an attack of influenza.

Pathological Anatomy.—There is catarrhal inflammation of the mucous membrane of the trachea and bronchi, and in the case of the finer tubes, of the bronchioles. This is associated with swelling and congestion of the mucosa, with, at first, diminution in the secretion of mucus, and later the pouring out of a copious and sometimes muco-purulent sputum, which is readily spat up by the patient.

Where the finer tubes are involved, small portions of lung collapse, and broncho-pneumonia frequently results.

On microscopic examination, in all forms of bronchitis there is found swelling of the submucous tissues and desquamation of the lining cells, which interferes greatly with the normal action of the ciliated epithelium of the bronchial tubes.

Affecting the Large Tubes, or Ordinary Acute Bronchitis

Clinical Features.—There is more or less pyrexia, with a temperature of 100° to 101° or 102° F., coming on after

exposure to cold or wet. During the *first stage* there is cough of an ineffective nature, because no mucous secretion is being poured out, and therefore there is nothing to be expectorated. There is severe pain behind the sternum, and a varying degree of general malaise and discomfort, and frequently pain in the back and legs.

During the *second stage* the patient coughs severely, but brings up a small quantity of scanty, and very viscid, mucus; after an expectoration there may be temporary freedom from cough. During both these stages there is apt to be a considerable degree of spasm, generally more marked at night, and causing dyspnoea in a severe case.

The *Physical Signs* of the two stages closely correspond. The breathing becomes harsh vesicular, with expiration prolonged, and the accompaniments consist of rhonchi, generally sonorous, sometime squeaking or sibilant. There may be some fremitus due to the rhonchi, and the percussion note is either unaltered, or is hyperresonant and suggestive of emphysema.

The *third stage* of bronchitis commences when the secretion becomes less viscid and often somewhat purulent; it is typically frothy, but comes up with great ease, and the cough is not so severe, and generally ceases temporarily after a fair amount of sputum has been got rid of. Rarely is the sputum tinged with blood, although not infrequently there may be particles of carbon intermixed with it. The only alteration in the physical signs during this stage is the addition of bubbling, moist râles, especially heard during inspiration and sometimes expiration.

In the other systems there is little to be noted; the loss of appetite and the frequency of constipation are common phenomena in any febrile affection, and there may be a scanty secretion of urine, precipitating a copious deposit of urates on standing.

A result of repeated attacks of bronchitis is the development of emphysema, with marked backward pressure affecting the right side of the heart, and a varying degree of dyspnoea resulting from it. The duration of an attack depends much on the personal equation of the patient, but an ordinary attack of bronchitis usually subsides in a matter of ten days to a fortnight.

The **Diagnosis** is easy, although it may be difficult to say

what the cause of the bronchitis is; and it is well to remember that a case of *measles, whooping-cough, or typhoid fever* may closely resemble a severe attack of bronchitis. The physical signs are definite, and should prevent confusion with cases in which consolidation is present.

Prognosis.—At the extremes of life bronchitis is always serious, and also in patients who are debilitated, or in whom the condition of the right heart or the degree of pre-existing emphysema affords reason for anxiety. It is invariably of grave importance when oedema of the bases of the lungs develops in a case of bronchitis, and if the inflammation spreads to the smaller tubes, the patient runs the additional risk of catarrhal pneumonia.

The **Complications** of bronchitis are emphysema, bronchorrhœa, bronchiectasis, cardiac failure from strain, especially upon the right heart, and the risk of broncho-pneumonia where the small tubes become involved.

Treatment.—The first stage of the disease should be treated by sedatives, and perhaps most effectively by the use of steam inhalations and the external application of linseed poultices, fly-blisters, or mustard. A good deal may be achieved by the administration of such remedies as antimonial wine (℥ 5-15), and ipecacuanha wine (℥ 15-40); while a purgative, together with a fever mixture, such as the spirit of nitrous ether, in drachm doses, affords the greatest and most prompt relief. The patient should certainly be kept in bed, until the temperature has fallen to normal.

During the *second stage*, when the secretion is viscid, much may be done to render it more easily expectorated, and potassium bicarbonate (gr. 5-15), and other alkalies, as well as small doses of the emetic group, such as apomorphine, are very serviceable, but are aided greatly in their action if poulticing or external counter-irritation be still kept up. A useful external rubefacient is the linimentum terebinthinæ aceticum, either applied alone or with an equal part of the linimentum saponis. Towards the end of this stage a stimulating expectorant, like ammonium carbonate (gr. 5-10), together with squill and senega, is often prescribed.

During the *third stage* an attempt should be made to lessen the secretion, and cough mixtures containing the mineral acids are specially useful for this purpose.

In many patients spasm predominates, and paroxysmal fits of coughing may cause great distress. The use of a tent over the bed into which steam is conducted, medicated with tincture of benzoin, terebene, creosote, or carbolic acid, is of great comfort to the sufferer, and apomorphine, given by the mouth or hypodermically, although it induces sickness, may relax the spasm and clear the bronchial tubes. Spirit of chloroform and tincture of hyoscyamus are also useful antispasmodics.

Spirit of chloroform, spirit of ether, and aromatic spirit of ammonia, together with alcohol in the form of whisky or brandy, are frequently necessary to support the patient's strength, and attention should be paid to the diet of the invalid during an attack.

For children a hot bath is an admirable method of commencing treatment, and where expectoration becomes difficult, an emetic, such as repeated large doses of ipecacuanha wine, clears the tubes at the same time as it empties the stomach of its contents.

Capillary Bronchitis, or Bronchitis affecting the Small Tubes

This form is common in children, and is generally secondary to fevers, and especially measles and whooping-cough. In adults, it is not infrequently due to the inhalation of irritating vapours, and is the precursor of septic pneumonia, which may arise from the inhalation of food particles, etc., in cases where the glottis cannot be closed, or in cases of cancer of the vocal cords. In patients suffering from excessive weakness, or where there is stricture of the bronchial tubes, often malignant in origin, the resultant septic pneumonia begins with a capillary bronchitis.

Clinical Features.—Should the capillary bronchitis produce, as it frequently does, a secondary catarrhal pneumonia, the reader is referred for a description of the latter condition to p. 426; but, so far as capillary bronchitis itself is concerned, there is invariably greater pyrexia than in bronchitis of the large tubes (104° to 105° or 106° F.). There is also more intense dyspnoea, more pain on coughing, and more rapid exhaustion; the pulse-rate may vary from 120 to 150; the respirations from 30 to 50, or even more, and the rapidity is

[illegible]

always greater in very young patients. The struggle for breath probably induces patches of the lung to become collapsed, and it has been stated that small areas of collapse form the stage between capillary bronchitis and catarrhal pneumonia, the patches themselves undergoing catarrhal inflammation. The proximity of the inflammation to the air vesicles greatly increases the patient's distress and dyspnoea, and there is always a grave depression of vital powers. Orthopnoea is invariably present, where the child is old enough and sufficiently conscious; the extraordinary muscles of respiration and the alæ nasi act with great vigour, while the lower interspaces tend to be sucked in with each inspiration.

Physical Signs. — On *Palpation* rhonchial fremitus, and sometimes fremitus due to moist râles, may be felt. The *Percussion* note varies considerably; it is dull, if consolidation or collapse ensues over any areas of lung so involved. *Auscultation* may reveal little definite change as regards the breath murmurs, excepting that they are weakened and sometimes absent, and the latter phenomenon may depend upon collapse or blocking of bronchial tubes. The accompaniments, however, are distinctive, numerous sibilant rhonchi and fine, moist râles being present over the greater part of the chest; and in old persons, in cases where the vital functions are becoming depressed, and where there is septic pneumonia setting in, these moist râles are very evident over the bases of the lungs.

The cough is often harassing, and may occur in severe paroxysms. The complications and sequelæ have already been noted; they are broncho-pneumonia, sometimes septic in nature, and collapse of the lung.

The **Diagnosis** is generally easy; but it may be difficult, except in the case of children, to recognise the cause of the condition, and an effort should invariably be made to eliminate — (1) a possible neoplasm involving one or other bronchus; (2) a laryngeal affection either paralytic or malignant; and (3) by investigation of the history the possibility of the inhalation of an irritant gas or of acrid fumes. The question of the failure of vital powers being responsible for the condition may also be considered, because debilitated alcoholic subjects are specially prone to capillary bronchitis of septic type.

The **Prognosis** is always grave where the small tubes are involved, although it may depend partly on the age and

strength of the patient. Extensive collapse and a definite catarrhal pneumonia increase the risks. The following clinical features are suggestive of grave danger:—(1) Where, in the case of a child, the dyspnoea is so great as to prevent crying^① and feeding, and where coughing, previously in evidence, has entirely ceased. (2) A great rise of temperature, or on the other hand a fall to 99° F., or even to normal, unaccompanied by improvement in the other features, generally precedes a fatal result. (3) When the child or adult becomes more markedly cyanosed, with a sudden and great increase in the dyspnoea, it implies, either that the heart is failing or that collapse has occurred; and (4) in children, the violent throwing about of the arms invariably portends an early fatal result. *Cyanosis is extremely present.*

The **Treatment** consists in endeavouring to support the patient's strength and to prevent pulmonary collapse occurring. It is exactly similar to that given for catarrhal pneumonia; but it should be remembered that the use of oxygen inhalations, the early and free exhibition of stimulants, alcoholic and other, and in children the induction of vomiting so as to clear the tubes, are of peculiar value in this type of bronchitis, as distinct from the involvement of the larger tubes. *Treat. Success possible (Pneumonia)*

(2) CHRONIC BRONCHITIS

A chronic inflammation of bronchial mucous membrane.

Etiology.—The *predisposing* factors are old age, a history of gout or rheumatism, alcoholism, Bright's disease, heart disease, certain cases of aortic aneurism, and chronic phthisis. *Michael &*

The *exciting* causes are cold and wet, as well as some of the above.

Pathological Anatomy. The bronchial mucosa is thickened, granular, and infiltrated, and ulceration is not infrequent. In other cases the mucous membrane becomes thin, and there is invariably a tendency for the muscular coat to yield to pressure, causing dilatation of the bronchial tubes, while the constant attacks of the disease lead to emphysema, generally bilateral, and involving sometimes the whole lung, although often most markedly the anterior margins. The glandular structures in connection with the bronchial tubes are frequently atrophied.

Clinical Features.—Typical attacks of chronic bronchitis may be seen in old persons during cold weather, and are frequently called “winter cough.” The appearance of the chest is that described under emphysema, and many of these patients have a typical barrel-shaped chest, while we get evidence of backward pressure due to the emphysema, causing dilatation and hypertrophy of the right heart, and, when compensation fails, dropsy of the extremities and all the phenomena which we associate with loss of compensation of the right heart.

The amount of cough depends on the severity of the attack of chronic bronchitis, while the degree of dyspnoea, in part due to the condition of the right heart and the emphysema present, varies also with the extent of the bronchial tubes involved and the quantity of secretion present in them. There is usually an element of spasm; some patients suffer from severe spasms of coughing and wheezing during the night, others have their worst attacks in the morning upon wakening, when the tubes are full of secretion, and are only emptied after a severe bout of coughing.

Physical Signs.—On *Inspection*, as already noted, the chest is typically emphysematous, and there may be evidence of backward pressure, such as the distended veins in the neck and the smaller venules seen over the chest wall. The extraordinary muscles of respiration are generally called into action during a severe attack. On *Palpation* the vocal fremitus is diminished, while fremitus due to rhonchi and moist râles is sometimes present, rhonchi being specially common during the earlier stages of the attack and moist râles during the later. The *Percussion* note is hyperresonant. On *Auscultation* the breath sounds are harsh vesicular, with expiration prolonged, the degree of prolongation depending on the element of spasm, which is always expiratory in type. The accompaniments during the early stages are sonorous rhonchi, and during the later stages bubbling, medium, and large moist râles, which are heard sometimes over the larger tubes alone and sometimes over the whole lung. In older and weaker persons moist râles of a finer character are extremely common at the bases, and indicate a degree of œdema of the lungs. The vocal resonance is slightly diminished, or may be unchanged.

The other physical signs depend on the condition of the

right heart and the presence or absence of tricuspid incompetence, with distension of the veins in the neck. Evidence of enlargement of the liver and spleen may be noted. Lastly, it should be mentioned that, as a result of extensive emphysema, the diaphragm is apt to be displaced downwards.

The **Complications** of chronic bronchitis, other than emphysema and enlargement of the right heart, depend on changes in the bronchial walls, which lead to bronchiectatic dilatations. Changes may also occur in the secretion contained in the bronchi, by which that secretion becomes excessive or foetid, or even gangrenous in odour.

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In Bronchorrhœa the sputum is excessive. It may be serous or mucous in character, and associated with marked œdema of the lungs, the patient spitting 2 or 3 pints a day, or it may be very purulent, when it tends to become foetid, and is generally associated with bronchiectatic dilatations. Foetid sputum implies infection with organisms, and it is easy to understand how ulceration may result in cavities containing such sputum, and how gangrene may ensue.

There is a mild form of chronic bronchitis in children, which may result in the same definite and permanent changes in the lungs and tubes as are associated with the disease in adults. It is, in reality, mild attacks of more acute bronchitis, from which the child may eventually completely recover when puberty is reached.

In older patients, there is sometimes a special form of chronic bronchitis in which no secretion occurs at all. This has been called dry catarrh, and it is invariably very resistant to treatment and is accompanied by marked emphysema.

Fibrinous or plastic bronchitis will be described separately.

The **Diagnosis** requires no reference, except the observation that it is wise to investigate the possibility of a tubercular element being present in cases which resist treatment.

The **Prognosis** depends much on the strength of the patient and the possibility of sending him to a warm climate during the winter weather. In every case, before an opinion is expressed, the condition of the right heart should receive special attention.

Treatment.—Of more importance than any drug is the question of climate. Bronchitic patients should be sent to warm, equable, and dry resorts, Egypt and the Riviera

being almost ideal during good winters. It is not judicious to send patients so affected to a high altitude unless the heart can stand the strain. Many bronchitic patients derive the greatest benefit from drinking a glass of warm milk and water, or a cup of weak tea, immediately upon wakening, because by this means the usual fit of morning coughing is rendered more easy.

The following drugs appear to be of special value:—

✓ potassium iodide in 10-15 grain doses, ammonium carbonate in 5-10 grain doses, and sometimes ammonium chloride, while liquor arsenicalis (℥ 5), continued for a considerable time, is often beneficial. Where the sputum is slightly foetid, terebene or guaiacol should be given in capsules (℥ 15-30). With the same object inhalations of antiseptics may be used, either vaporising and inhaling carbolic acid, creosote, or guaiacol, or having in the sick-room and beside the patient's bed a porcelain capsule, in which some volatile antiseptic, such as one of those just mentioned, may be slowly vaporised.

Much good may follow external counter-irritation, and the linimentum terebinthinæ aceticum, equal parts of turpentine and olive oil, and many other rubefacients, are of great value. Where backward pressure is excessive, and the patient capable of standing it, bleeding may be thought of, and, lastly, the treatment given for emphysema is often applicable in cases of chronic bronchitis.

Liniments

(3) FIBRINOUS OR PLASTIC BRONCHITIS

An acute or subacute form of bronchitis, in which fibrinous casts of the bronchial tubes occur, and by their presence cause violent paroxysms of coughing.

Etiology.—Casts of the bronchi form in diphtheria, when the membrane extends down into the bronchi; sometimes in pneumonia and phthisis similar casts may develop. It has been stated that after the inhalation of ammonia, alcohol and eau de Cologne, after the local application of lactic acid, and in some rare cases after the exhibition of potassium iodide, fibrinous casts may be formed in the course of a bronchitis which has otherwise nothing very peculiar in connection with it.

Clinical Features.—Paroxysmal attacks of coughing

occur, after which the patient expectorates one or more of these casts generally with much mucus and blood; the casts may be formed in the larger or smaller tubes; they may, therefore, be of large or of small size, and usually are markedly branched. They are hollow casts made of fibrin or mucin, and contain in addition many leucocytes, alveolar epithelial cells, frequently Charcot-Leyden crystals, and sometimes Curschmann's spirals. There is great dyspnoea associated with the disease, which, however, is relieved after the separation and expectoration of the casts. *not in it*
sampled

The **Diagnosis** depends on the recognition of the casts in the sputum.

The **Prognosis** is bad in diphtheritic cases, and this type of bronchitis is always a tedious and severe one.

Treatment.—The casts are soluble in lime-water, and Dixon has suggested the use of this in an atomiser; a solution of papain, which is said to have similar solvent properties, may also be tried. Emetics are not infrequently of value, and sometimes inhalations of ether have been advised. The disease is rare, and therefore the treatment must depend a good deal on the individual case. *of difference in bronchitis*

(4) BRONCHIECTASIS; DILATATION OF BRONCHIAL TUBES

It may be general or saccular, and it is due either to weakening of the bronchial wall, traction upon it, or increased intrabronchial pressure.

Etiology. 1. One of the common causes of bronchiectasis is weakening of the bronchial wall as the result of chronic inflammation, and especially in bronchitis. The muscular coat and the elastic tissue in the bronchial wall both yield, and associated with this weakening, due to long-continued inflammation, there is also the important factor of intrathoracic pressure to be taken into account. Cough implies the closure of the glottis and sudden and powerful contraction of the expiratory muscles; therefore in cases where the bronchial wall is weakened, such pressure, if frequently brought into play, will result in dilatation of the tubes as a whole, or more likely of those parts where the resistance is weakest. In addition, however, to weakening of the wall and increased intrathoracic pressure there is a third factor present in certain cases,

namely, the accumulation of secretion, which by its retention will tend to produce or increase local dilatations.

It is easy to understand how interference, such as might be caused by stricture of a bronchus from tumour, gumma, or other condition, will result in time in localised dilatation of the distal bronchial tubes, because it is largely the result of retained secretion.

2. Traction upon the bronchial walls is exercised by thickened septa in the lung, and especially where this condition attains a marked degree. In fibroid lung, and particularly that type associated with chronic tuberculosis, such traction may produce local dilatations, and it seems probable that pleuritic adhesions will aid in the process, because they give a definite fixed point from which traction may be exerted.

3. There is a rare congenital form of bronchiectasis, due to universal weakening of the bronchial walls, generally limited to one lung. It seems probable that, in still more exceptional cases, such general dilatation of tubes may be met with in the adult, but it must imply an embryonic error in the structure of the bronchi.

Pathological Anatomy.—Saccular dilatations are certainly more common; they have smooth walls, unless ulcerative changes, from retained secretion or tubercular processes, are present. The muscular coat of the bronchus is atrophied, the elastic tissue weakened, and the fibres widely separated from one another. The shape of the cavity depends much upon the amount of traction. In some cases there may be saccular dilatations throughout the greater part of one lung, or part of a lung; in other cases one or two saccular dilatations may alone be present, associated with tubercular or other fibroid changes, and these are very common near the apices of the upper lobes. Surrounding the dilatations, there is generally some increase of fibrous tissue, but the amount depends largely on the nature of the case.

The **Clinical Features** vary according to whether the cavities are saccular, or whether the dilatation is more universal. There is generally the expectoration of large quantities of sputum, clearing the bronchiectatic cavities, and thus relief from the cough is obtained. These fits of coughing may be invariably looked for in the morning, when the patient awakens.

The sputum tends to be foetid in odour, and when placed in a glass vessel separates into three layers, of which that on the top is brownish, frothy, and muco-purulent; the middle layer is clear, mucus-looking fluid, and in the foot of the glass there is a thick deposit, granular and yellowish in colour. This deposit is largely made up of pus cells, of fatty crystals, bacteria, and often elastic tissue of the lung, where there is ulceration. Hæmorrhage is not an uncommon phenomenon in this disease. There is a varying amount of dyspnœa depending much on interference with the lungs.

The cylindrical or general form of bronchiectasis closely resembles chronic bronchitis, as regards the physical signs and clinical features, while in the saccular form, the physical signs are those of a cavity, and include the cracked-pot note on percussion, cavernous or amphoric breath murmurs, consonating moist râles, and whispering pectoriloquy. There are very often evidences of fibroid changes in the lung, such as local retraction of the chest wall and the displacement of neighbouring organs, so commonly the result of such changes.

Complications.—There are four important complications or sequelæ:—(1) the development of gangrene, (2) the not infrequent onset of severe hæmorrhage, (3) the occasional association of rheumatoid joint pains with the disease, and (4) in rare cases, the metastatic cerebral abscess or abscesses which are secondary to the pulmonary condition. In addition it is only necessary to remark that fibroid lung throws great strain on the right heart.

Diagnosis.—In many cases the condition is merely suggested by the clinical features and physical signs, and accurate diagnosis is impossible. In practically every case of fibroid lung, saccular cavities are met with, and it is hardly possible to discriminate between the vomicæ of chronic tuberculosis and bronchiectatic dilatations. There is frequently clubbing of finger-tips and marked dyspnœa on exertion, in cases of bronchiectasis.

Prognosis.—Recovery is rare, but the condition is compatible with considerable prolongation of life. There is always the risk of septic absorption, and specially so where the contents of the cavities undergo decomposition changes.

The Treatment is not satisfactory. We may try to render the contents of the cavities a little less septic, but this is by

no means easy. Probably the best intrathoracic injection consists of guaiacol 2 parts, menthol 10 parts, in 88 parts of olive oil, and a drachm of this solution may be injected twice daily. It is impossible to ensure that this or any other injection should reach the affected part of the lung, but it is very difficult to adopt any other method. Direct injections of solutions of iodoform into the cavity through the chest wall are both painful and not very desirable, because a septic needle track, at least, on the removal of the syringe may give rise to much trouble. Inhalations of creosote, guaiacol, turpentine, thymol, and carbolic acid may be used in many different ways, both volatilised and by spray, but care should be taken that the patient does not suffer from toxic doses of the drug used.

Every effort should be made to support the general strength, and to endeavour by patient medical treatment eventually to diminish the excessive secretion in the bronchiectatic dilatations.

The surgical treatment of bronchiectatic cavities is eminently unsatisfactory, unless adhesions have occurred between the layers of pleuræ, and even then the risk is very great.

(5) ASTHMA

Recurrent attacks of expiratory dyspnœa, often extreme, and generally nocturnal.

Etiology.—It is undoubtedly hereditary, and especially common in neurotic families. It may follow bronchitis or whooping-cough in those who have a constitutional predisposition; gout and possibly syphilis are responsible for some cases. It occurs at all ages, more frequently in males.

The exciting causes of an attack are many—peripheral irritants, hay pollen, nasal polypi, adenoids, dyspepsia, mental irritation, fright and many other conditions of a varied nature.

Pathology and Pathological Anatomy.—That asthma is due to spasm of bronchial tubes is beyond question, but there is much difference of opinion as to how this is brought about. There is frequently also catarrh of the mucosa associated with the condition. Charcot-Leyden crystals and Curschmann's spirals are not limited to the sputum of asthmatic

patients, and they may have no causal connection. A vaso-motor paresis is a favourite theory, but there is nothing to prove its existence.

The anatomical changes present are those met with in bronchitis, or are mostly sequelæ such as emphysema; while an eosinophilia, usually present in the blood, does not offer any solution to the problem of causation.

Clinical Features.—There are often premonitory phenomena, such as mental irritability, and then the attack develops. The patient awakens at 1 or 2 A.M. with a sense of great oppression and huge efforts at inspiration, while expiration is prolonged, wheezing, and difficult. He becomes cyanosed, and beads of sweat appear on his face, which wears an anxious expression. The spasm yields after a period of time, and during the attack the patient usually coughs up some viscid sputum containing the peculiar casts of small bronchial tubes called Curschmann's spirals. The attack generally recurs the following night, while during the day the patient may be fairly comfortable.

Physical Signs.—The chest is fixed at nearly full inspiration, and the inspiratory effort demands the aid of the extraordinary muscles of inspiration; the appearance of the chest closely resembles the type in emphysema, with the lower interspaces widely separated and indrawn on inspiration, while the chest as a whole is hoisted up rather than expanded. The shoulders are rounded and stoop, and there may be actual spinal curvature. On *Palpation* there is usually rhonchial fremitus. On *Percussion* a hyperresonant note is obtained, while it is found that the superficial cardiac dulness has disappeared, and that the absolute liver dulness is much diminished. On *Auscultation* there is harsh vesicular breathing with great prolongation of the expiratory murmur, and the accompaniments are sibilant, rarely sonorous, rhonchi, and at later stages there may be moist râles. There is no pyrexia as a rule.

Diagnosis.—Cardiac and renal asthma are types of dyspnœa which have kindred characteristics, but they do not give the complete clinical picture of ordinary bronchial asthma, while in each case the recognition of cardiac or renal disease should help to solve the difficulties of diagnosis.

The **Prognosis** is good in children, in whom the disease may be arrested at, or by, puberty, but in adults the resultant

changes in lungs and heart (emphysema and enlargement of right heart) render the outlook less favourable.

The **Treatment** of the attack consists in antispasmodics, nitrite of amyl, in 3 minim capsules, broken in the handkerchief and inhaled, or nitroglycerine in doses of $\frac{1}{100}$ th to $\frac{1}{25}$ th grain; inhaling the vapours from burning stramonium, datula tatula, and nitre paper; and in certain cases with great caution the use of chloroform anæsthesia, or morphia (gr. $\frac{1}{8}$ - $\frac{1}{4}$) with atropine (gr. $\frac{1}{20}$ - $\frac{1}{80}$), are excellent remedies. Chloral, bromides, and many other sedatives and hypnotics may also be administered.

Much can be done to obviate the attacks by trying to remove any of the known exciting causes, such as dyspepsia, hay pollen, and so forth, and every case demands a careful dietary with a sufficiency, but no excess, of easily digested food, and the regulation of the meal hours, so that no heavy meal is taken towards bedtime. Warm clothing should be recommended, and, if possible, the patient should be advised to live, during the cold and damp months of the year, in a mild, dry climate. Inhaling compressed air is often beneficial, and big doses of potassium iodide are serviceable in some cases.

The neurotic element when present should not be forgotten, and suitable tonics should be prescribed.

IV. PULMONARY EMPHYSEMA

THE pathological term for abnormal dilatation of pulmonary alveoli and air vesicles.

The theories and the common varieties of emphysema form an excellent introduction to a study of the subject.

The *Theories* are three in number:—

(1) Primary weakness or degeneration of the air-vesicle walls.

(2) An inspiratory theory of production, the air not getting out, and each fresh inspiration distending the air vesicles still more (Gairdner). (*Kaennel*)

(3) An expiratory theory: cough and excessive expiratory effort certainly produce emphysema.

Probably a combination of (3) and (1) explains most cases of emphysema.

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There are three *Varieties* of emphysema:—

- (1) A general type, often known as the large-lung type of emphysema.
- (2) An atrophic or small-lung type, the result of atrophy, and met with in old age.
- (3) Compensatory emphysema—where emphysema occurs in the same lung, or perhaps in the opposite lung, as the result of local interference with part of a lung.

Etiology.—*Age.*—It may occur at any age—the atrophic form in old persons. *Sex.*—It is commoner in men. *Trades.*—It is met with in persons who blow wind instruments which tax the expiratory powers, such as the cornet, in glass-blowing, and in all occupations which tend to induce bronchitis. *Disease.*—Bronchitis and whooping-cough unquestionably cause emphysema.

Whether there is a hereditary tendency is doubtful; certainly it seems to run in certain families.

Pathological Anatomy.—The lung affected is typically dry and bloodless; the air vesicles run together, with atrophy of the walls and obliteration of the capillaries. The anterior margins and apices of the lungs suffer most, but compensatory emphysema may occur in any part of the lung. There is often considerable atheroma in the larger branches of the pulmonary artery. Large bullæ sometimes develop under the pleura, and there may be interstitial emphysema in the lung substance. The pathological results of emphysema include enlargement of the right heart, with evidence of chronic venous congestion in the liver, the kidneys, and the venous circulation generally. Pneumothorax may result from rupture of one of the bullæ referred to.

The **Clinical Features** of type (1), or *General Emphysema*. There is dyspnœa, especially on exertion, stooping, and even, in marked cases, produced by talking. It may be so severe that the patient has to sit up in bed (orthopnœa). Cough depending on bronchitis is often present. There is more or less cyanosis, and the veins in the neck may be engorged.

Physical Signs.—*Inspection.*—The chest is very characteristic. It is barrel-shaped, with considerable increase in the antero-posterior diameter, the chest being fixed in the position of nearly full inspiration. The epigastric angle is abnormally obtuse, the lower interspaces widened, and the upper narrowed.

The shoulders stoop, there is often actual spinal curvature, the supraclavicular fossæ are deep, and the neck appears short, with well-developed sterno-mastoid, scalene, and the other muscles of extraordinary inspiration. The sternum is unduly prominent, and on inspiration there is little or no expansion, but the chest is hoisted up as a whole, while the diaphragm contracts vigorously. There is a deep furrow marking the usual upper transverse abdominal line. Frequently venous radicles are seen on the chest, and especially along the line of insertion of the diaphragm. There is marked bulging in the supraclavicular regions on coughing.

Palpation reveals that the vocal fremitus is diminished.

On *Percussion* the note is (hyperresonant) and there is usually no superficial cardiac dulness. The heart, and especially the liver, are displaced downwards. *Auscultation* reveals feeble breathing, unless bronchitis is present, with prolongation of the expiratory murmur. The vocal resonance is diminished, and in the absence of bronchitis or œdema there are no accompaniments.

Very rarely serious hæmoptysis occurs, and death is generally due to cardiac or pulmonary complications.

(2) In the *Senile* or *Small-Lung Type* of emphysema, the chest is small, the sternum thrown forward, and the apices flattened. The note on percussion is hyperresonant, and the absolute cardiac dulness may or may not be diminished. There is little or no displacement of heart and liver.

Diagnosis.—It is almost impossible to mistake a well-marked case of general emphysema, while compensatory emphysema, being a common result of any interference with an area of lung, hardly demands much reference. The apices and anterior margins should always be examined in cases where emphysema is probably present.

The **Prognosis** of the general form is unsatisfactory. It is usually progressive, and if so, leads to ever-increasing interference with the circulation.

Treatment.—Prevent the condition from becoming worse. Treat the bronchitis, induce the patient to seek a warm, dry climate in winter, and keep up the general health. Stop any effort which throws great strain on the air vesicles; in other words, prevent forced respiratory efforts. Tonics, such as iron, strychnine, and especially arsenic, are of value; try also to

relieve the backward pressure by diuretics and purgatives. Much benefit may be obtained by placing the patient in a pneumatic chamber, where he breathes condensed air, usually at a pressure of $1\frac{2}{3}$ atmospheres; expiring into rarefied air has also been commended, but is difficult to manage. Pulmonary gymnastics, which help in expressing air out of the chest during the act of expiration, are certainly of value.

V. HÆMOPTYSIS

(PULMONARY HÆMORRHAGE)

HÆMOPTYSIS is a sign of many pulmonary and other diseases, rather than a separate disease in itself.

Etiology.—Hæmorrhage may occur from the nose or from the trachea, or one of the big bronchi, but it is hæmorrhage from the lung with which we have specially to deal.

(1) It is certainly most common in cases of phthisis, in connection with which disease it is described elsewhere.

(2) It is a common result of pulmonary congestion, and is seen in acute lobar pneumonia and in lobular pneumonia, although in these conditions the amount of hæmorrhage is not, as a rule, great.

(3) Hæmoptysis may be a sequel to pulmonary abscesses of all kinds, and specially where gangrene is present, and also to pulmonary ulceration, such as occurs in stone-mason's lung. It is a common and important clinical feature in cancer of the lung.

(4) Hæmorrhage is one of the clinical features of pulmonary infarction; the amount of blood expectorated varies greatly, and generally continues for some days.

(5) In the rare instances of the involvement of the bronchial tubes by the *Distomum Westermanni*, considerable hæmorrhage may occur.

(6) Rupture of an aortic aneurism may take place into the lung or into a bronchus, and cause death from fatal hæmorrhage.

(7) In a group of cases where, probably, there is little acute congestion of the lung, hæmoptysis may occur: these include severe strain, as from lifting a heavy weight, and the

hæmoptysis sometimes associated with the commencement of a continued fever; it also occurs as the result of backward pressure, as in common cirrhosis of the liver, and cases of backward pressure with engorgement of the pulmonary circulation.

(8) In certain blood diseases, such as purpura hæmorrhagica, anæmia, hæmophilia, and scorbutus, hæmorrhage may take place.

(9) Injury to the chest frequently causes hæmoptysis.

Vicarious menstruation is a rare cause of hæmoptysis; and in a few cases a trivial hæmorrhage has occurred, in elderly persons, probably from the rupture of pulmonary vessels whose walls have become degenerated.

Pathological Anatomy.—Blood in the lung undergoes the usual decomposition changes, and, therefore, if a hæmorrhage has taken place, the patient will go on for some time expectorating sputum containing blood which has become altered in this way; the rusty sputum in pneumonia is a good example.

In tubercular disease of the lung hæmorrhage often occurs, not only from the rupture of a vessel in a cavity, but from small miliary aneurisms formed in tubercular cavities. In tubercular cases a hæmorrhage is often followed by a rapid spread of the disease.

Clinical Features.—The bleeding may be excessive or slight; it may be long continued, or there may be only one considerable attack of hæmorrhage, lasting a comparatively short time.

A peculiar saltish taste in the mouth, sometimes with, sometimes without, cough, is generally the first indication to the patient that blood has entered the mouth, while a feeling of tightness, or even of pain, referred to the chest, is very common. The blood is bright and frothy, very different from the dark, partially digested blood which comes from the stomach. Clots may be coughed up, and the amount of blood depends on the cause of the hæmorrhage. There is generally, in a severe hæmorrhage, great palpitation and a feeling of faintness, the extremities become cold and clammy, and beads of perspiration appear on the forehead.

Very frequently careful examination of the chest with the stethoscope enables the physician to recognise the site of the hæmorrhage, but the greatest care should be taken to avoid

moving the patient, and so risking a renewal of the hæmoptysis. The breathing over the area of hæmorrhage may be somewhat bronchial in character, but the distinctive feature is the presence of fine or medium moist râles.

Diagnosis.—Blood from the *stomach* is generally dark and partially digested, and gives an acid reaction, while blood from the lung is bright red and alkaline. It must be remembered, however, that in hæmorrhage from the lung some of the blood may be swallowed and then vomited. There may be a feeling of pain or tightness over the chest, suggesting that the lung is the source of the hæmorrhage, whilst the bright, frothy blood is very typical. Bleeding from the *nose* is generally easily differentiated.

Prognosis.—Whether the hæmorrhage is severe or slight, it is prudent to assure the patient of the fact that he is in no immediate danger, because death rarely occurs as an immediate result of hæmoptysis. It is hardly necessary to add, however, that where an aneurism of the arch of the aorta has ruptured, or where a miliary aneurism in the lung in a case of phthisis has given way, a fatal result is by no means uncommon, and it may occur with startling suddenness.

Treatment.—Where the hæmorrhage is slight, it may be only necessary to confine the patient to bed, to remove the pillows, to loosen any garments round the neck, and to enjoin absolute repose for a considerable period of time. An ice-bag applied over the chest, and, if possible, over the site of the hæmorrhage, is of great value, and small pieces of ice to suck, while they may not avail greatly, give the patient something to do—a point of much importance in relieving his anxiety.

A hæmorrhage frequently ceases when syncope occurs, the pressure in the vessels being then greatly lowered, and to aid in this lowering of pressure, aconite and other remedies are sometimes administered. It is certain that opium, given in the form of the liquor morphinæ (℥ 10-20), not merely soothes an excited heart, but helps also to allay the patient's anxiety. Styptics of different kinds should be administered, such as turpentine, in 15-30 minim doses in capsules, mineral acids, and especially dilute sulphuric acid (℥ 10-15), tincture of the perchloride of iron (℥ 20-40), gallic acid (gr. 10), acetate of lead (gr. 1-2), tincture of hamamelis (℥ 30-60), and chloride

of calcium in doses of 10 to 20 grains three or four times daily. Some authorities have suggested that where the hæmorrhage is continuous, and the patient does not faint, an attempt may be made to reduce the pressure in the pulmonary circulation by bleeding from the arm. This method is certainly open to considerable objection.

For a few days after the hæmorrhage has occurred the patient should be confined to bed, and it must be kept in mind that a marked degree of pyrexia may follow, especially in tubercular cases; the food must be as simple as possible, and no alcohol should be allowed. All strain, especially physical, must be interdicted, and the lungs should be overhauled from time to time, so as to find out whether there has been complete absorption of the effused blood, because there is always a risk that a hæmorrhage may induce rapid spread of pulmonary tuberculosis, should it be associated with that disease.

VI. ACUTE PULMONARY CONGESTION

AN active congestion of the lung and a condition similar to the first stage of lobar pneumonia.

Etiology.—It may be caused by the inhalation of irritating vapours or very hot air, and it may result from excessive action of the heart as in fevers or overstrain. In a localised form it is associated with many lung diseases, such as pneumonia, phthisis, and so forth.

The **Pathological Anatomy** is simply active engorgement of the lung, general or local, with blood.

Clinical Features.—There is a varying degree of pyrexia, 102° F. or more, sometimes with hæmoptysis. The physical signs are rather negative than positive. There is defective expansion, feeble breath murmurs, and diminished resonance. Otherwise the condition shows the characters of the disease of which, as a rule, it forms a part.

VII. PASSIVE PULMONARY CONGESTION

THIS specially involves the lower lobes, and there is usually some œdematous effusion into the air vesicles.

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Two types demand a brief description:—

1. *Hypostatic Congestion* is the result of gravity plus debility. The affected lung is of a dark purple colour, and is engorged with blood and œdematous fluid, and may even sink in water. The dependent parts alone suffer.

Clinical Features.—There is local dulness varying in degree, with typical moist râles and generally feeble bronchial breathing. There is always dyspnœa, and a low type of inflammation may supervene, which rapidly kills the patient.

The **Prognosis** is grave, and the **Treatment** consists in stimulation both by internal remedies, such as alcohol, ammonia, and spirits of chloroform, and also locally by poulticing and even counter-irritation of a more vigorous nature. Try to avoid the onset of this condition by altering the patient's position in bed from time to time.

2. *Backward Pressure Congestion* is the pathological condition called brown induration of the lungs, showing distended, tortuous, alveolar capillaries, with much coloration, with blood pigment, of the alveolar epithelial cells. There may be blood or blood pigment in the sputum.

This type follows many valvular lesions of the heart. There is usually a considerable amount of œdema present.

The **Clinical Features** vary greatly and depend on the amount of œdematous exudation into the air vesicles.

The **Diagnosis** may therefore be rather a question of inference in milder cases.

The **Prognosis** depends on the causal condition, and the **Treatment** is directed to the relief of the backward pressure by kidneys and bowels, possibly by cardiac tonics, and in some cases by direct blood-letting, when 20-30 oz. may be taken from the arm.

VIII. PULMONARY ŒDEMA

THIS is present to a very limited extent in active or acute congestion, but it is common in passive pulmonary congestion, and there is a so-called acute idiopathic variety, such as occurs in Bright's disease, and after the administration of pilocarpine, to which reference will be made later.

In ordinary pulmonary œdema the lung is full of water.

fluid which may be expressed post mortem from the cut section, and is coughed up during life in large quantities by the patient. It is greatest in the dependent parts of the lungs.

The **Clinical Features** include those of the heart disease or other causal condition, but also very marked dyspnœa, with cough and watery sputum. There are two reliable physical signs, namely, the dull note, sometimes with a tympanitic note over the neighbouring lung, and the moist râles of small size, very freely heard on coughing.

The ultimate **Prognosis** is never or rarely favourable, and the **Treatment** consists in counter-irritation locally, free internal stimulation with alcohol, ammonia, and similar remedies, and an attempt at alleviating the conditions to which the œdema is due. Propping the patient up in bed is of special value.

Acute idiopathic œdema, not an uncommon cause of death in Bright's disease, may come on very rapidly; the clinical features correspond to backward-pressure œdema, only the cause of the backward pressure with its characteristic features is wanting.

IX. CATARRHAL PNEUMONIA

(BRONCHO-PNEUMONIA; LOBULAR PNEUMONIA)

A CATARRHAL inflammation of localised, generally multiple, portions of the lung immediately consequent on capillary bronchitis.

Etiology.—(1) There is a form of broncho-pneumonia which is a lobular variety of lobar pneumonia, and which appears to be rather more frequently met with in children when subject to the attack of the pneumococcus.

(2) A large proportion of cases of broncho-pneumonia are, however, secondary to a capillary bronchitis. Many of these originate in an attack of whooping-cough, measles, influenza, or some other continued fever in children, and certainly the death-rate in measles and whooping-cough is not so much due to the primary disease itself, as to the broncho-pneumonia to which it may give rise. It is specially common under the age of five, and most fatal in children who have not yet reached their third year. The incidence of the broncho-pneumonia

may depend partly on the time of year and partly on the debility of the patient.

In adults a broncho-pneumonia, resulting from a capillary bronchitis, is often associated with Bright's disease and any condition in which the vitality of the patient has seriously suffered; while in diphtheria at all ages the membrane may infect the smaller tubes, and so give rise to a broncho-pneumonia of diphtheritic origin.

(3) There is yet another type of broncho-pneumonia which may be divided into two groups:—(a) In one, where there is paralysis of laryngeal muscles, food may be inspired, and a deglutition pneumonia results. A similar state of things follows in certain cases where the patient is comatose, or where there is paralysis of the vagi nerves, or in any condition in which a septic pneumonia may be set up by the inspiration of foetid mucus or of food particles. Purulent discharge in connection with the ulcerations due to malignant disease, and, in certain cases, the filling of the lung with blood, may both cause a septic pneumonia.

(b) The other subdivision of this group of broncho-pneumonias is the form which is associated with excessive weakness, and which often is a condition immediately preceding death. In it, the mucus is not got rid of, and tends to decompose and to act as an irritant. This mucus is drawn farther into the smaller tubes with each inspiration, and may there set up a condition of things akin to a septic pneumonia. The debility is greatly aided by alcoholic tendencies in the patient, and death generally occurs before there are many patches of lobular pneumonia actually in existence.

The tubercle bacillus most frequently attacks the lung so as to set up a tubercular broncho-pneumonia, but this specialised form of the disease is described separately under pulmonary phthisis.

Pathological Anatomy.—The inflammation begins in connection with the little bronchus leading to a lobule, and is often preceded by the collapse of the lobule in which the consolidation is about to occur. The central bronchus is filled with leucocytes and swollen, granular epithelial cells; the bronchial wall is inflamed, and the surrounding air vesicles are filled with leucocytes, epithelial cells, red blood corpuscles, and a proportionately small amount of fibrinous exudation. At the

post-mortem these pneumonic patches stand out prominently as small projections of the lung, and the pleura covering the patches shows fibrinous exudation. On section the patches are reddish-grey in colour, and the lobules in the neighbourhood are often collapsed. Minute hæmorrhages may occur under the pleura, and the areas of broncho-pneumonia may be seen, in many different positions, in the lung. In septic cases they are often met with in the lower parts of the lung; in children, where it is a sequel to measles or whooping-cough, they may be widely scattered over both lungs. The patches frequently resolve, but they may lead to suppuration or even gangrene, or may undergo a fibroid change.

In septic cases the affected bronchi contain little plugs of

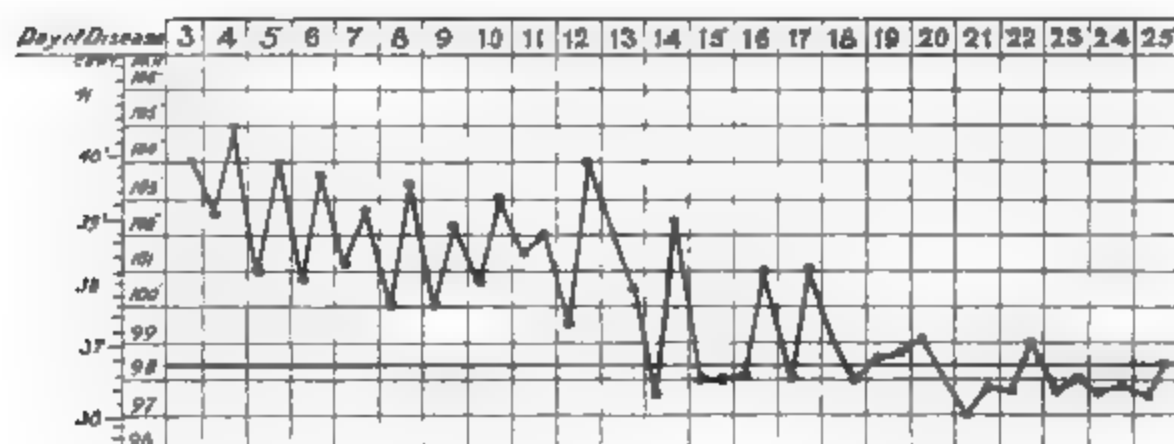


DIAGRAM 26. Temperature Chart. Broncho-pneumonia. Recovery.

pus, and the small lobular areas of hepatisation are seen to be undergoing softening.

Bacteriology.—In addition to the *Pneumococcus* of Fränkel and the *Pneumo-bacillus* of Friedländer, there are many organisms which may be found in broncho-pneumonia; these are specially the *Streptococcus pyogenes*, the *Staphylococcus aureus* and *albus*, sometimes the *Diphtheria bacillus*, and, as already indicated, the *Tubercle bacillus*.

Clinical Features.—*Type 1.*—This type is simply the lobular form of lobar pneumonia; it corresponds in physical signs and clinical features to lobar pneumonia with, in addition, the characteristics of type 2, about to be described.

Type 2.—In a child who has had whooping-cough or measles, broncho-pneumonia may set in with severe rigors, or the temperature may gradually rise higher and higher until it reaches 105° or 106° F., the dyspnoea becoming much greater, the child markedly cyanosed, and the lower interspaces more

and more sucked in. As broncho-pneumonia follows the antecedent capillary bronchitis, the child gradually becomes more seriously ill, and increase of the patient's distress usually indicates a rapid spread of the disease to a larger number of lobules of the lung. The pulse-rate may rise, in a child, to 150 or 200, and the respirations may reach 60 or 80 per minute; the face has a peculiarly anxious expression, and the cough, paroxysmal and exhausting, becomes less and less noticeable as the patient reaches the stage of stupor.

On examining the chest, we find evidences of small areas of consolidation which yield the phenomena already described under capillary bronchitis. As these areas are by no means always superficial, it may be difficult to elicit evidence of their presence by percussion. The breathing over the areas becomes tubular, and, what is even more typical, fine moist râles, sharp and clicking in character, are heard over the patches. The phenomena are similar to what is noted in capillary bronchitis, but they are associated with a greater degree of distress. The condition may last for 1, 2, or 3 weeks, and then it will either terminate favourably or death may occur. The temperature falls in most cases by lysis; only in those patients in whom the broncho-pneumonia is the result of the pneumococcus or the pneumo-bacillus does the temperature sometimes fall by crisis.

Type 3.—In septic broncho-pneumonia, where due to the inspiration of food, pressure on the bronchus, or other cause, the areas of consolidation are found in the lower parts of the lung. The dyspnoea is usually less extreme, the patient is in most cases an adult, and the disease follows a more chronic course. There may be increasing evidences of septic absorption, but very frequently the condition is rather suspected than actually diagnosed during life.

There are obviously cases of broncho-pneumonia which do not correspond closely to any of the types just mentioned, but sufficient has been said to make the leading clinical features of the condition recognisable.

Leucocytosis is frequently present, excepting in post-influenzal cases.

Diagnosis.—A careful examination should be made of the sputum so as to search for the presence of the pneumococcus, and it should be remembered that in children who are the

subjects of a pneumococcal pneumonia, the distribution of the disease is apt to be lobular rather than lobar. The close association between measles, whooping-cough, and influenza and catarrhal pneumonia is often of great help in diagnosis, and a capillary bronchitis, from which the little patient does not show signs of recovering, generally implies the onset of catarrhal pneumonia. In such a case the greatly increased dyspnoea and cyanosis, with the indrawing of the lower interspaces, should make the physician extremely suspicious.

Typhoid fever may, at its commencement, show symptoms rather suggestive of a capillary bronchitis than of the typical phenomena of the fever, and in doubtful cases the Widal reaction should be tested, though it should be remembered that at least a week must elapse after typhoid fever has commenced, before the reaction may prove positive.

Many cases of *acute broncho-pneumonic tuberculosis* closely resemble broncho-pneumonia, but it seems hardly necessary to remind the reader that the discovery of the tubercle bacillus in the sputum is the one certain method of diagnosis. In *miliary tuberculosis* of the lungs, there are usually no lobular areas of dulness, and the tubular breathing generally got over such patches, does not occur. In addition, the moist râles in miliary tuberculosis are not, as a rule, clicking in character. Unfortunately, it is extremely common to find miliary tuberculosis associated with broncho-pneumonic tuberculosis, but in such a case the tubercle bacillus will sooner or later be obtainable in the sputum.

Tubercular meningitis sometimes closely resembles broncho-pneumonia in its general features, but the rigidity of the neck with the drawing back of the head, and the tendency, eventually, for the involvement of cranial nerves, render the diagnosis of basal meningitis fairly clear. Lumbar puncture may also aid the physician, as the cerebro-spinal fluid often contains an excess of leucocytes and an increased proportion of albumin.

Prognosis.—The death-rate from broncho-pneumonia is very high in children, but much depends on the constitution and age of the patient; probably over 50 per cent of all cases terminate fatally in patients under the age of three. In the lobular form of pneumococcal pneumonia, the prognosis is favourable. It is hardly necessary to add that septic pneumonias of all kinds are very apt to prove fatal, and that

where an irritant substance of any kind has been inhaled, the death-rate must be high.

Treatment.—The utmost care should be taken of children suffering from the fevers to which capillary bronchitis and broncho-pneumonia are such common sequelæ. The child's strength should be carefully maintained, and every care taken to avoid chill and fatigue during convalescence from the fever in question. When it is evident that capillary bronchitis has set in, the main object of the physician should be:—*Firstly*, To give the patient moist air to breathe at a fairly warm temperature, and this is best done by covering in the top of the bed or crib with a "tent," into which a bronchitis kettle discharges steam; *Secondly*, to endeavour to prevent the bronchial tubes from being filled up with mucus, by encouraging coughing, and, when necessary, giving an emetic to clear the tubes. Much benefit results from altering the position of the patient in bed, keeping the head high, and from time to time lifting the little patient, so that he may get change of position together with some stimulation.

Thirdly, Alkalies should be administered for the purpose of diminishing the viscidty of the sputum, and stimulants, such as aromatic spirit of ammonia, spirit of chloroform, and spirit of ether, will also be found valuable. Alcohol has an important place among the necessary remedies. The local application of stimulating liniments, such as camphorated oil, or an occasional linseed poultice, is excellent, and will often help in enabling the patient to clear the bronchi of secretion. A jacket poultice is good practice, but requires the greatest care in its application, because it readily exhausts the patient's strength, and must not, therefore, be used too frequently. The application of ice, or the ice-water jacket, may be useful in cases where the child's strength is well maintained; but it is doubtful whether such treatment is prudent as a general rule. *Fourthly*, Perhaps there is no disease, with the exception of lobar pneumonia, in which oxygen inhalations prove such a valuable aid to the physician at a critical time. *Fifthly*, and lastly, Remember to keep a constant and careful watch on the temperature; it is probably wise to instruct those in attendance that the temperature should be kept *below* 103° F., and that, whenever it tends to rise to 102.5° , cold sponging, or the use of the cold bath, or other similar means for lowering

the temperature, should be promptly resorted to. The number of cases of broncho-pneumonia which are lost because of the neglect of this precaution, is very considerable.

It seems hardly necessary to refer in detail to the diet; it should be of the simplest, the child being fed with milk and beef-tea or egg-flip every two or three hours; and it is wise to procure free, but not excessive, action of the bowels, to help the kidneys in the discharge of their functions, and to endeavour to ensure for the patient as much sleep during the night as possible. Probably one of the first and best signs that the disease is yielding to treatment, is the appearance of moisture on the skin coincident with a steady, but gradual, fall in temperature. For long after an attack of broncho-pneumonia the child's health and general well-being demand careful consideration.

X. PULMONARY COLLAPSE

COLLAPSE of portions of the lung may occur either from the pressure of pleural effusions, from occlusion of a bronchus, or from some difficulty in the way of the air entering part of the lung (commonly found in the lower parts of the lung).

The **Etiology** of pleural effusions is described under the appropriate heading. Bronchial occlusion may result from the presence of a diphtheritic membrane, excessive bronchial secretion, especially in children, or the pressure of a tumour upon the bronchi. The form of collapse associated with the lower parts of the lung is a frequent result of excessive debility, and generally occurs shortly before death. In cases of great cardiac enlargement collapse of the lower part of the left lung is by no means uncommon.

Pathological Anatomy.—The portion of lung collapsed is dry and airless; it is somewhat tough when cut with a knife, and is of a bluish-grey colour.

Clinical Features.—Where a large area of lung collapses, dyspnoea results, and may be associated with great cyanosis, which tends to increase until the death of the patient. The changes depend largely on the amount of lung affected; local flattening and immobility of the affected part of the chest are typical; the percussion note is dull, although the degree of

dulness varies with the amount of lung involved; the breath murmurs are absent or greatly enfeebled, and the vocal resonance is much diminished. In certain cases it should be remembered that the collapsed lung may conduct breath murmurs better than normally, so that distinct bronchial breathing, with bronchophony, may be sometimes noted.¹

Diagnosis.—The dyspnoea, together with the immobility of the affected part of the chest, and the physical signs just mentioned, are diagnostic of collapse, but, on the other hand, the collapse noted after death, in debilitated patients, may give rise to little definite clinical phenomena.

The **Prognosis** is grave; it is only in cases where small areas of collapse are associated with broncho-pneumonia that a favourable result may be anticipated. Serious collapse occurring in young children in connection with capillary bronchitis or broncho-pneumonia is of very grave import.

The **Treatment** should consist in free stimulation, generally by the administration of alcohol and such drugs as ammonia, strychnine, etc., while the application of mustard poultices and counter-irritation with stimulating liniments should be persevered with. In young children, collapse is frequently due to an excessive amount of secretion blocking the bronchial tubes. Encouraging the child to cough, or giving him emetics, may tide him over a serious condition. When the stomach is emptied, the bronchial tubes are also cleared, but emetics are depressing, and therefore stimulation must afterwards be vigorously carried out.

ATELECTASIS

Deficient expansion of the lungs at birth. It is sometimes the result of feebleness, and where expansion does not occur, the child remains cyanosed, and generally soon dies, sometimes with convulsive seizures. The usual treatment is to stimulate the surface of the skin by the alternate application of cold and hot water, and in most cases this treatment is alone sufficient to effect full expansion. The trachea or bronchi may be blocked with secretion, and it is easy, by mechanical means, sometimes by stimulating the child to cough or making him sick, to clear away the obstruction.

¹ This depends on the bronchus remaining patent. Collapse with tubular breathing and bronchophony is not uncommon near the left base in cases where an enlarged heart or distended pericardial sac compresses the lung.

XI. PULMONARY TUBERCULOSIS

(1) ACUTE TUBERCULOSIS OF THE LUNGS

(a) Type 1. Lobar Pneumonic Tuberculosis

THIS is a rare form, and it is not always easy to ascertain by what channels the whole of one lobe has become involved. There are generally a few caseous foci which probably indicate an older disease, but the most of one lung, or one or more lobes, are consolidated and closely resemble the grey hepatisation stage in an ordinary lobar pneumonia. On careful examination with the magnifying glass, numerous tubercle nodules are seen dotted in miliary arrangement throughout the whole of the consolidated lung, although it is quite probable that, in certain cases, the disease may be a combined attack by the tubercle bacillus, together with one or other of the organisms responsible for croupous pneumonia. There is generally some pleurisy, and tubercle nodules may be dotted over the pleura.

The **Clinical Features** begin with a sudden rise of temperature, with rigors, great dyspnœa, and pleuritic pain associated with cough, while the sputum is not very dissimilar to that in croupous pneumonia, being generally tenacious and blood-stained or rusty in appearance. There may be the typical malar flush, so commonly seen in croupous pneumonia, but soon after the period when the crisis should arrive, there is obvious evidence of destruction of lung tissue, and the sputum generally becomes extremely purulent, and on examination is found to contain numberless tubercle bacilli. The temperature swings, often reaching 103° or 104° F. at night, and falling to about 101° during the day, with considerable sweating; this tends to increase as the disease progresses.

The *Physical Signs* are those of lobar pneumonia, with, in addition, evidence of rapid excavation, and the breathing associated with this disease is just as typically tubular as in lobar pneumonia. Death occurs with great rapidity, often in 2 weeks, sometimes 4 to 6 weeks, but very rarely does the disease become chronic. Sometimes, in cases which probably are in reality a mixture of true pneumonia in addition to the tubercular element, the consolidation may largely clear up, but

it leaves a part of the lung evidently the site of tubercular disease.

Diagnosis.—There is a close resemblance between this condition and *lobar pneumonia*, only the temperature swings more in the tubercular form than in the other, and the presence of tubercle bacilli in the sputum, with early evidence of breaking down in the lung, as shown by the appearance of elastic tissue in the sputum, renders the diagnosis easy, at least after a certain length of time has elapsed.

(b) *Type 2. Acute Broncho-pneumonic Tuberculosis*

This is another form of an extremely acute invasion of the lung by the tubercle bacillus. Probably in these cases the tubercle bacilli are inhaled, and we find the grape-like clusters corresponding to the distribution of the bronchial tubes. Round about the affected bronchial tubes there is a definite catarrhal pneumonic process, and in very acute cases the little caseous areas are apt to run together to form much larger masses. The lung between these masses is congested, and the pleura may show tuberculous pleurisy. Generally these areas are more marked in the upper lobe and near the apex, although any part of both lungs may be involved. Sometimes there is evidence of a much older focus of tubercular disease, from which the recent acute broncho-pneumonic involvement has originated. Cavity formation early occurs, and not infrequently the larynx and trachea are found to be affected.

Clinical Features.—The onset is sudden, starting with rigors, much like Type 1; but there are cases where an insidious commencement ushers in the attack. Not infrequently it is secondary to influenza. Sometimes after an insidious commencement, a considerable hæmoptysis precedes the rapid development of the disease. There is loss of appetite, the tongue is furred, and there is vomiting; the sputum is mucopurulent, and sometimes blood-stained, and later it becomes markedly purulent. There are many tubercle bacilli, and there is much elastic tissue in the sputum; the temperature usually reaches at least 103°, possibly 104° F., and the clinical features soon demonstrate the rapidly advancing and serious nature of the disease, vomiting and diarrhoea being common

symptoms. A good deal depends on whether the process is wide-spread, or limited to one lobe or one lung; where there are many scattered areas of involvement, the results of septic absorption are much more pronounced.

The *Physical Signs* are those of small patches of consolidation, which, however, may run together to form larger and more extensive areas. *Inspection* may show diminished movement, very probably well marked at, or near, one or other of the apices. *Palpation* reveals increased vocal fremitus, and also the fremitus of accompaniments which are generally medium and coarse moist râles. On *Percussion* the areas of dulness are sometimes easily mapped out, and where excavation has commenced, a tympanitic or cracked-pot note may be obtained.

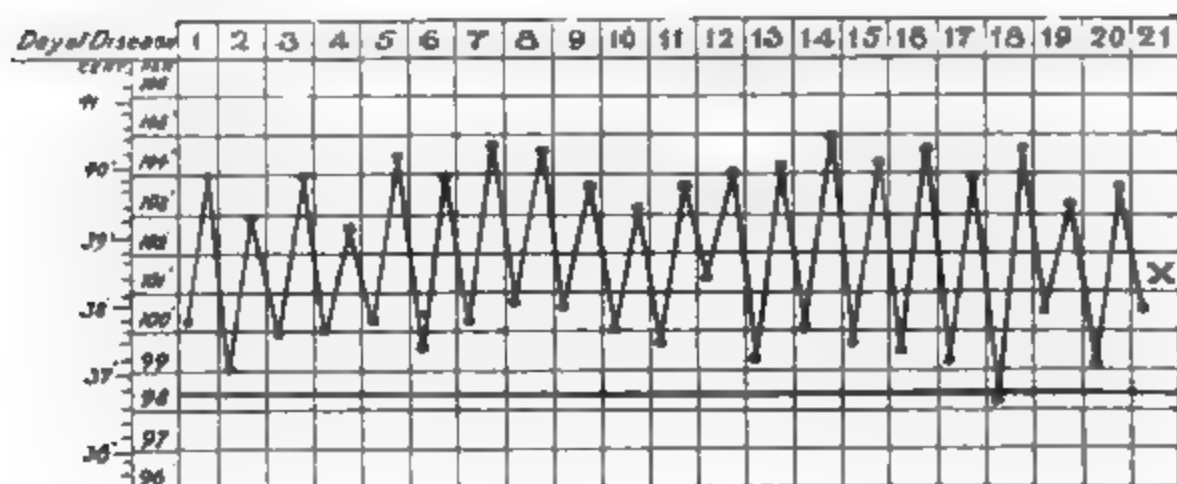


DIAGRAM 27.—Temperature Chart. Pulmonary Tuberculosis. Hectic temperature; fatal termination.

Auscultation yields harsh vesicular breathing, with expiration prolonged, often of a bronchial character, while over patches of consolidation the breath sounds are frankly bronchial. Over the cavities it is cavernous, but one important feature of the breath murmurs in tubercular disease is the jerky or cog-wheel character, so frequently made out over less advanced areas of the disease. This jerky character does not disappear when the patient takes a deep breath. In addition there are numerous clicking moist sounds specially heard during inspiration, and which are increased on coughing. Should excavation be progressing, these moist râles become consonating. Vocal resonance is usually bronchophonic, and whispering pectoriloquy is often obtained over the cavities. The presence of friction is not always easily made out, owing to the existence of so many moist râles, but the friction sounds are not altered by coughing; they increase in distinctness when firm pressure

is made with the chest piece of the stethoscope, and they are generally accompanied by pain, of which the patient usually complains. Friction is also, as a rule, better marked at the *end* of inspiration and the *beginning* of expiration, and becomes much more distinct when a deep breath is taken. The sputum usually contains many tubercle bacilli and much elastic tissue, although it must be remembered that death may occur before the disease has reached the stage of extensive lung destruction.

Diagnosis.—The rapid emaciation of the patient, the evidences of the spreading consolidation in the lung, extending not infrequently with alarming rapidity, and the typical temperature with hectic sweating, render the diagnosis easy even in young children who may swallow the sputum.

The **Prognosis** is always grave, and death may occur nearly as rapidly as in Type 1. Fortunately the disease can be arrested, and many months may elapse before death occurs, or the condition may pass into the chronic stage, although with a lung very seriously damaged.

(c) *Type 3. Miliary Tuberculosis of the Lungs*

Pulmonary tuberculosis of the miliary type may be part of a general involvement, or it may be limited to one lung, or merely part of a lung. Very probably a limited miliary tuberculosis is the result of ulceration through the wall of a pulmonary artery of one or more tubercle nodules, and thus the blood-stream to the part of the lung supplied by the artery in question may become infected by tubercle bacilli. The nodules are scattered in the line of the vascular distribution, and not in the line of the peribronchial lymphatic system; and along with this condition there is usually a varying degree of pulmonary congestion, and sometimes consolidation.

Clinical Features.—Where there is extensive involvement of the lungs, the phenomena met with in general miliary tuberculosis will be present. The attack begins with rigors, there is generally headache and malaise and marked loss of appetite, the face may become flushed, and the temperature varies from 102° to 105° F., falling towards the early morning, with profuse sweating. There is a short, dry, hacking cough, and the respirations are generally from 50-80 per minute,

while the patient usually shows marked dyspnoea, sometimes with cyano-sis. The sputum is often clear mucus, sometimes it is muco-purulent, and occasionally blood-stained.

The *Physical Signs* are chiefly negative; unless there is some patch of older tubercular disease, there may be nothing distinctive on inspection, palpation, percussion, or auscultation, excepting the presence of a few moist râles. In some cases we find a trace of albumin in the urine where tubercle nodules are deposited in the kidneys, and tubercles in the alimentary tract are not infrequently associated with marked changes. Often vomiting, sometimes diarrhoea, and occasionally waxy degeneration, are present, and in certain cases definite tubercular ulceration in the lower part of the ileum. In the *Hæmopoietic System* the spleen is often enlarged, with sometimes

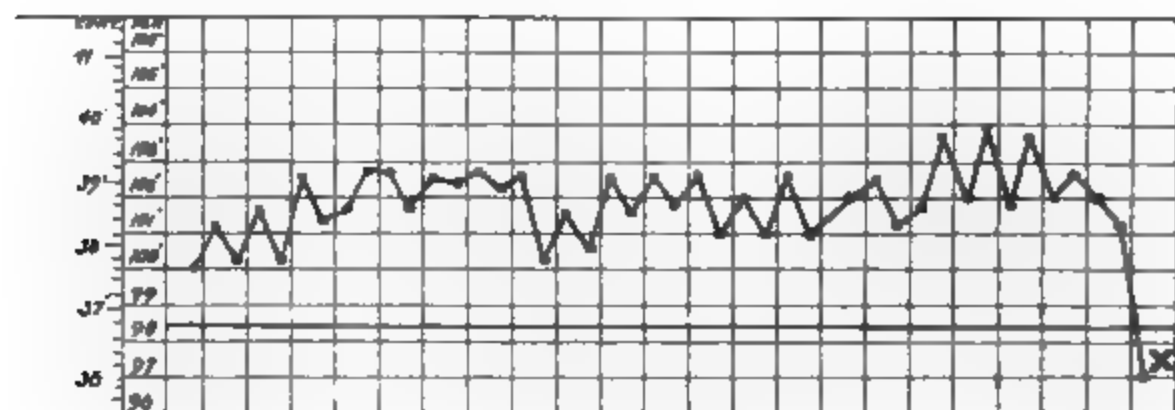


DIAGRAM 28.—Temperature Chart. Miliary Tuberculosis of Lungs; fatal termination.

the presence of miliary tubercles in the organ. In the *Circulatory System* there is nothing distinctive of the disease, but in the *Nervous System* there may be tubercular meningitis, and sometimes the appearance of miliary tubercles in the choroidal coat of the eyes. The clinical features of tubercular meningitis are described under the diseases of the Nervous System (p. 599).

The patient usually sinks early into the typhoid state, and the phenomena are generally governed by the degree of involvement of the meninges.

The **Diagnosis** is by no means easy; the possibility of *typhoid fever* should invariably be kept before the mind, and Widal's reaction is of great value in some of these doubtful cases. It should be remembered that in many cases of miliary tuberculosis there are no tubercle bacilli in the sputum, and unfortunately tubercles in the choroid are rather the exception than the rule.

In *acute capillary bronchitis*, advancing to *catarrhal pneumonia*, there is sometimes doubt as to whether the case is not really one of miliary tuberculosis. Generally a few days will clear up the diagnosis, because the definite areas of dulness in catarrhal pneumonia are not present in miliary tuberculosis, and as a rule the temperature is less hectic in non-tubercular catarrhal pneumonia than in a miliary tuberculosis, although the temperature chart on the opposite page shows only slight hectic characteristics.

Prognosis.—It is rare for the patient to survive more than 3 or 4 weeks, but sometimes life is prolonged for 3 to 4 months, and occasionally miliary tubercles become obsolete, and the patient recovers. Unfortunately this latter course is very exceptional.

The **Treatment** must be on general lines, and nothing need be said as to particular methods likely to yield good results. If any points demanded special mention, they would be the value of oxygen inhalations, and the benefit which may be obtained from the application of cold to the chest wall.

(2) CHRONIC TUBERCULOSIS OF THE LUNG: CHRONIC PULMONARY TUBERCULOSIS

The majority of cases of pulmonary phthisis may be grouped under this heading, and for purposes of description an early case of the disease will be briefly considered in the first place, and afterwards the typical features of one more advanced.

Etiology.—There is little to be said with regard to the etiology of this form of tuberculosis that does not apply to the other forms just considered. There is, in the first place, a very important hereditary predisposition, a predisposition which rather implies insufficient development of the chest and a want of vitality of the individual as a whole, than the direct transmission from either parent of the tubercle bacillus. The types of chest associated with tubercular disease are either—(1) *alar*, in which, along with anterior flattening, there is always marked prominence of the wings of the scapulae posteriorly, or (2) the chest may be *flattened*, with sloping shoulders and a distinct cervical stoop almost amounting to spinal curvature.

With these peculiarities there is sometimes a long, hollowed-out sternum, with a narrow subcostal angle. In the subjects so affected there are often evidences of rickets with the knob-like prominences at the junction of rib and cartilage, and a very marked indrawing of the chest, commonly known as Harrison's sulcus, which is just above the attachment of the diaphragm on either side, and which slopes downwards from near the sternum towards the mid-axillary line. In many of these cases there is bad nutrition, sometimes a history of very insufficient feeding and care, and not infrequently also inherent delicacy. Unquestionably the presence of adenoids neglected in youth may lead to the kind of defective development of the chest to which reference is made.

The disease generally manifests itself about the age of adolescence, or just after puberty, when probably the strain of life is greatest. In persons with a tubercular predisposition, exposure to cold or wet, sleeping in an ill-aired apartment, working at a dusty occupation, the strain of profuse menstruation, some debilitating illness, and especially influenza, even a great mental worry—these, and similar, causes may induce an attack of pulmonary tuberculosis. Reference has already been made in previous pages to the virulence of tuberculous sputum, and to the risk of tubercle bacilli becoming disseminated throughout the air of rooms or houses, and, given a suitable nidus, there is the greatest risk of the tubercle bacillus, inspired into the lung, setting up active disease. It is a remarkable fact that in asylums and other institutions in which, for various reasons, free life in the open air is not possible for all the inmates, tubercular disease of the lungs is extremely common.

Pathological Anatomy.—In most cases pulmonary tuberculosis is met with first near the apex of the upper lobe of one lung, and it seems probable that this seat of election is due to the fact that there is less exchange of air in that portion of the lungs. A not infrequent position is the apex of the lower lobe, although this is generally a secondary involvement, the original focus being in the upper lobe. It is not the *extreme* apex of the upper lobe which is usually the first part invaded, but it is about an inch below the extreme apex. The right apex is slightly more frequently involved than the left, but in a large proportion of cases both apices are found to

be affected. The distribution of the disease is in the line of the peribronchial lymphatics; it is therefore a chronic broncho-pneumonic tuberculosis, and the tubercle bacilli, once they have entered the lung, are distributed largely by the lymph-stream, although it is not uncommon to find, as the result of the invasion of the wall of one of the branches of the pulmonary artery, that a limited miliary tuberculosis is also present. The cavities found in the affected lung are the result of excavation of caseous areas. These are closely related to the bronchial tubes, and the cavities are lined with a pyogenic membrane often dotted over with miliary tubercles. The cavities have not got rounded walls as a rule, but are irregular, one cavity sometimes appearing to have numerous branches corresponding to the divisions of the original bronchi. Most large cavities have septa running through them in which blood-vessels pass. These vessels are generally obliterated, as the result of endarteritis obliterans, but sometimes they remain patent, and are invaded by tubercle bacilli, as already mentioned, and occasionally these form fairly large, aneurismal dilations which may nearly fill the whole of the cavities in which they develop. Such aneurisms not infrequently give way, and may lead to fatal hæmorrhage. The amount of destruction of the lung varies greatly; sometimes the whole of one lobe may be excavated, in other cases the cavities are of small size, and the old bronchial walls can be readily recognised. Coexistent with the more chronic varieties of the disease there is marked interstitial change—a fibrosis of the lung, which is the commonest process of cure of a tubercular lung. The degree of this fibroid change varies greatly; it may be very marked and very extensive; it may, in other cases, be comparatively limited, and associated with it there is almost always more or less thickening of the pleura, sometimes with pleural effusions, and often with the presence of tubercle nodules on the pleura. Not infrequently, where the disease becomes quiescent at a very early age, calcareous deposition occurs in caseous nodules, together with a limited fibroid change such as we have just described, but it is extremely rare to find, even after many years of quiescence, that the tubercle bacilli have absolutely and finally disappeared. The glands at the root of affected lungs generally show caseous foci, and in these caseous areas calcareous

deposition is extremely common, although sometimes the glands soften and form small abscesses.

Amongst changes in other organs to which chronic tuberculosis, primarily in the lungs, may give rise, the following may be mentioned:—In cases where there is much suppuration, waxy degeneration of the liver, spleen, kidneys, and alimentary tract may ensue, leading to the rapid death of the patient. In other cases tubercular ulceration of the intestine may follow from the swallowing of tuberculous sputum, and as a secondary result the mesenteric glands may become affected. The larynx, frequently involved by a comparatively mild catarrh of the vocal cords, sometimes shows marked phthisis laryngea, but far less frequently than one would expect. In connection with the circulatory system there is occasionally tubercular pericarditis and endocarditis, and it is only necessary to mention the fact that miliary tuberculosis or tubercular infection of other organs of the body may be a sequel to pulmonary tuberculosis.

Clinical Features of a Case of Early Pulmonary Tuberculosis.—There are many modes of onset of the disease. In not a few cases it begins insidiously, and only at a later date is attention directed to the lungs. In a second group of cases a hæmoptysis, which may be extremely severe, is the first indication to the patient of any pulmonary mischief; in another group of cases a severe bronchitis occurs, from which the patient does not completely recover. Pleurisy is yet another affection which appears to usher in pulmonary tuberculosis; there may or may not be effusion, but the cough from which the patient suffers, remains behind, even after the effusion and friction have disappeared. It is an important point to remember that in young patients apparently suffering from anæmia, or from some alimentary disturbance, the real cause of the trouble may be pulmonary tuberculosis.

While it is true that the shape of the patient's chest, with the presence of cough and sputum, may yield important clues to the diagnosis of an early tuberculosis, there are only two or three clinical features specially typical of pulmonary tuberculosis at a very early stage:—

(1) The respiratory capacity of the lung in pulmonary tuberculosis always falls far short of the normal amount, and were it possible to use the spirometer, the information

yielded would be of great value. A careful note, however, should be made of even the slightest difference of movement between the two sides of the chest, although it is true that in right-handed persons the right side of a normal chest acts more vigorously than the left, and that sometimes from rickets or other cause the usual symmetry of the chest may be altered, and that such alteration may have little connection with phthisis pulmonalis.

(2) The breathing over a patch of early phthisis is generally cogwheel-like or wavy, and this peculiar character is not lost when the patient breathes deeply.

(3) There is generally some secretion, even although the amount of sputum may be extremely small, and a few clicking sounds are often detected over the affected lung when the patient takes a deep breath, and especially after coughing.

The Clinical Features of a more advanced Case of Pulmonary Tuberculosis.—Pain in the chest and cough are two early and important features; the pain is often pleuritic, and may occur over an affected apex, or may be referred to one shoulder blade. The cough is generally distinctive; it is short and dry, with, at first, little expectoration, and indeed the appearance of much secretion is dependent on the presence of bronchitis or the commencement of disintegration processes in the lung. The cough frequently induces vomiting, and perhaps especially when it has a laryngeal as well as a pulmonary cause. It is a remarkable fact that although serious involvement of the larynx is by no means common in pulmonary tuberculosis, catarrh or simple anæmia of the laryngeal mucosa is associated with between 80 and 90 per cent of all cases.

The sputum is typical; it may vary from muco-purulent to purulent, it may be nummular, and it may, or may not, contain blood, but sooner or later, in every case of chronic broncho-pneumonic tuberculosis, tubercle bacilli will be found, and in very many cases, elastic tissue from the air-vesicle walls is readily recognised. Tubercle bacilli should be stained by the Ziehl-Neelsen method described on p. 116. The amount of sputum varies greatly; it may be foetid, or merely have the peculiar sweetish odour so characteristic of tuberculous sputum. Sometimes blood appears, either in large or in small amount. It is very rare to find that a case of tuberculosis runs its whole course without any hæmoptysis, but

only in about half of the cases does the spitting of blood become an important feature. Such hæmoptysis may be the result of severe strain, possibly merely the stress of coughing; in other cases it is due to rapid destructive processes in the lung; while in yet others, and those are cases of very severe hæmorrhage, it may result from the rupture of a miliary aneurism in one of the cavities. It is very rare indeed to have a severe hæmorrhage in the early stages of phthisis, although in the later stages the bleeding is apt to be more copious. Where there has been a considerable hæmoptysis, blood is expectorated for several days afterwards, and clots and blood-casts of the bronchi may be coughed up. Sometimes old calcareous masses are found in the sputum. These indicate calcareous deposits in an old caseous nodule, and their

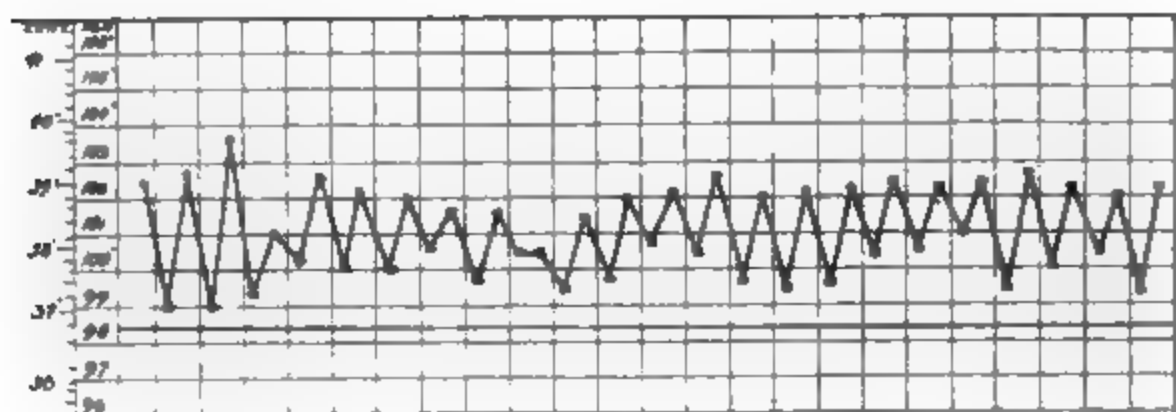


DIAGRAM 29 —Temperature Chart Cuscuting Pulmonary Tuberculosis. Advancing Disease.

appearance is generally the result of a local recrudescence of the disease permitting these little nodules to be separated and spat up in the sputum.

Dyspnoea depends largely upon the area of lung involved, and whereas, in some cases, breathlessness may merely follow from exertion, in other cases it is exceptionally severe, and is associated with more or less cyanosis.

Emaciation is usually marked, and the sharply cut features, so frequently noted in phthisical patients, depend largely on loss of flesh. The temperature varies within wide limits; in mild cases a nocturnal rise of 1° or 2° may be the limit of the pyrexia, whereas in other cases the temperature may reach 102°, 103°, or 104° F., generally falling towards morning with excessive sweating. The temperature chart is often a useful guide in deciding whether the tubercular process in the lungs is extending, because a minimum temperature which

never reaches the normal has been described by the term *remittent*, and generally indicates advancing disease (see Chart opposite). On the other hand, a daily variation of temperature of 2° or 3° is not incompatible with quiescence of a tubercular area. Where there is a tremendous fall, reaching normal or below normal, and associated with pronounced sweating, the temperature is described as *hectic*, and implies septic absorption, and not infrequently indicates an early and fatal termination of the illness (see Chart on p. 436). Improvement in the patient's condition in the direction of quiescence is usually demonstrated in the temperature chart by a lessened degree of pyrexia at night, and as a result diminished sweating. The perspirations, so typical of the phthisical patient, frequently saturate the night-dress, and render the use of flannel or woollen garments imperative. The sweating is to a large extent the result of toxins produced by the tubercle bacillus. There is almost always more or less malaise, the degree depending largely on the activity of the pulmonary disease.

Physical Signs associated with the Respiratory System.—*Inspection* generally reveals one or other of the types of phthisical chest, and the flattening, with marked lagging, of the affected apex or base is usually evident. The expansion of the affected part of the lung is invariably deficient, and the utmost care should be taken to compare accurately the movements of the two apices, as viewed by looking over the patient's shoulders, so as to contrast the one side with the other. *Palpation* greatly aids in appreciating diminished expansion, and it is well to take a measurement of the amount of expansion of both sides of the chest on full inspiration. The lagging, referred to under inspection, is more obvious to the hand on palpation. The vocal fremitus is generally increased if the area of disease enables the vibrations to be better conducted. The *Percussion* note over the affected lung is dull, and should be very carefully compared with the corresponding area on the other side; and not merely will the ear enable the observer to obtain a fairly accurate impression of the amount of consolidation, but the sense of resistance to the hammer finger is greatly increased. A tympanitic note is got over a superficial cavity, and when the patient opens the mouth and breathes deeply, the typical cracked-pot note is obtained during expiration. Cavities are most frequently to be found just below the clavicles

and also at the apices of the lower lobes. *Auscultation.*—The breath sounds vary greatly in different cases; they may be faint, from interference with the entrance of air into the affected part of the lung; they may be harsh vesicular with expiration prolonged, or they may be frankly bronchial; the inspiratory murmur is sometimes cogwheel-like, and where cavities are present the bronchial breath sounds acquire a cavernous, or possibly amphoric, character. Tubular breathing is not so common as in the acute lobar pneumonic type of the disease. The accompaniments, generally best marked after coughing, have a peculiar clicking character, and they may be fine, medium, or large in size, while in cavities they acquire consonating qualities. Rhonchi are frequently met with, depending, however, on the amount of bronchitis present. The vocal resonance is increased and may be bronchophonic, and whispering pectoriloquy is obtained over cavities. Pleuritic friction is often present.

Cavities are generally easily diagnosed: the tympanitic or cracked-pot note, the cavernous or amphoric breathing, the consonating moist râles (should secretion be present in or near the vomica), and lastly, whispering pectoriloquy, are all typical phenomena. It should, however, be remembered that a cavity may be full of secretion, in which case the typical signs will only be obtained when, after a fit of coughing, it has been emptied of sputum. Cavities may also be deep-seated, and therefore may not yield a definite tympanitic or cracked-pot note on percussion. It is necessary to point out that the size of a cavity cannot be gauged by the pitch of the tympanitic note obtained, because the quality of note depends greatly on the size of the opening of the bronchial tube into the cavity. The larger the opening, the higher in pitch does the note become, and the opposite also holds good, that the smaller the opening, the lower in pitch is the note on percussion. The coin test is applicable to large cavities, and can be used for the definition of their boundaries. It consists in tapping one coin on another coin placed on the chest wall over the cavity, while with the stethoscope, also placed over the cavity, the ringing sound obtained is conducted to the ear.

Very frequently there is evidence of displacement of neighbouring organs, and especially the heart, as the result of tubercular disease in the lung. This is common in cases

where fibroid phthisis has developed, the heart being pulled over toward the affected side.

Complications and Sequelæ referable to the Respiratory System.

—The *larynx* is apt to be involved in a considerable percentage of cases; not merely the slight huskiness of voice already mentioned, but definite tubercular ulceration may occur. There may also be many miliary tubercles scattered over the mucosa of the trachea and larger bronchi, and there is always a tendency, where a tubercular area of lung exists, to find that the disease spreads by neighbouring and communicating bronchi to other parts of the same lobe or the same lung. This is probably due in many cases to sputum overflowing into a neighbouring bronchus, along which it may be inspired, and such an accident is peculiarly liable to occur where there is much sputum, and when it gets a chance of accumulating during sleep. Hæmorrhage is also a common mode of extension of the disease, blood, containing tubercle bacilli, being not infrequently inspired into healthy lung, the hæmorrhage having occurred in the tubercular area.

Emphysema is an almost inevitable sequel in cases of tubercular disease of the lung. It affects the healthier portions of the diseased lung, and sometimes the whole or the greater part of the opposite lung. In reality it is a compensatory process, and unless very marked, rarely causes much, if any, trouble to the patient.

Pneumothorax is one of the most serious sequelæ to which a phthisical patient is prone. Where a cavity near the pleura ulcerates into the pleura, a pneumothorax results, possibly limited by adhesions, and possibly involving the whole of the pleural sac. The patient becomes very dyspnœic, and death is hastened. Not infrequently a pyopneumothorax develops and adds much to the distress of the patient.

Pleurisy has already been described, and is a part of the tubercular process; but it may be mentioned that effusions are not uncommon, sometimes ordinary inflammatory lymph, and sometimes a purulent effusion. Dry pleurisies, limited, as a rule, to the neighbourhood of the disease, are very common.

In a few cases of advanced pulmonary tuberculosis the lung becomes the site of invasion by *gangrene-producing organisms*, and death may follow, under conditions peculiarly trying for the patient and attendants alike.

A certain proportion of cases die from pneumonic involvement of the lung, sometimes infection with the pneumococcus, and sometimes the development of acute pneumonic tuberculosis which has already been described.

Clinical Features associated with other Systems.

Alimentary System — Dyspepsia is an almost constant feature in marked pulmonary tuberculosis, and not infrequently vomiting and diarrhoea ensue, the former perhaps largely due to the stress of coughing, the latter often the result of tubercular ulceration or waxy degeneration of the intestinal tract. The tongue is very typical, it is furred and generally moist, but in severe cases it may be covered with patches of thrush, which are also seen on the gums and fauces. Where there is phthisis laryngea, pain on swallowing is very common, and adds greatly to the difficulty of feeding the patient.

Hæmopoietic System — Anæmia is frequent, but beyond the fact that a leucocytosis generally develops where there is much pus formation, the blood yields little useful information.

Circulatory System.—The displacement of the heart and the retraction of the anterior edge of the left lung are common features in cases in which there is much cirrhosis. The pulse is generally of low tension and often markedly dirotic.

Integumentary System. The skin of a phthisical patient may be typical; the hair is apt to become thin and dry, pigmentation of the skin may occur, and many phthisical patients show very marked freckling. It is common to find the presence of pityriasis versicolor on chest and back, due to the presence of the fungus, *microsporon furfur*, and probably largely induced by the excessive sweating. Very rarely do the patches cause itching or give any trouble. The tips of the fingers and toes may show very marked clubbing, peculiarly evident in emaciated patients, and usually associated with extensive fibroid phthisis.

Urinary System. The appearance of albumin in the urine may be significant of a secondary involvement of the kidneys by miliary tubercle or of waxy degeneration, more frequently it is simply due to backward pressure.

Nervous System — Many patients suffering from pulmonary phthisis develop tubercular meningitis. A much more un-

usual condition, and probably really the result of alcohol, or possibly some drug, such as arsenic, is the appearance of peripheral neuritis in the later stages of pulmonary phthisis. A curious feature of many phthisical patients is the happy, hopeful spirit to which the term *spes phthisica* has been applied. It not merely implies that the patient is bright and cheerful, but includes a strong expectation on his part of final recovery, and the worse the disease, the more hopeful may he become.

A familiar phenomenon called myoidema may be noted on percussing the chest; the portion of intercostal muscle contracts after tapping, and remains so contracted, forming a local swelling for a considerable period of time. This phenomenon should more correctly be described under the locomotory system; its significance is unknown.

Course, and Mode of Termination of the Disease.—A case of pulmonary phthisis may be chronic from the outset, and may, at any stage, become quiescent, the affected parts of the lung being gradually surrounded by fibrous tissue. In other cases the disease may slowly progress, the patient living in comparative comfort for many years. In yet other cases the disease becomes fibroid phthisis, tubercular in nature, but owing to excessive suppuration it may finally kill the patient, either from septic absorption or waxy degeneration, or possibly simply from exhaustion. Chronic pulmonary tuberculosis presents itself to the physician under many different forms and types, and much experience is necessary in order to recognise the exact position of matters in the lungs and the probable course of the disease.

Not a few cases die of sheer exhaustion; others, as already noted, die of pneumothorax; others from severe pulmonary hæmorrhage, or from the development of gangrene: and probably in a larger number of cases than our present knowledge suggests, death may be due, in part, to thrombosis occurring in important branches of the pulmonary artery.

Diagnosis.—There is no disease which, in an advanced stage, is so easily recognised as pulmonary tuberculosis. In most cases tubercle bacilli and elastic tissue are found in the sputum, and even where there is no cough and perhaps no sputum, the physical signs of tubercular consolidation are definite and typical. They may for a time be confused with

catarrhal pneumonia, but the patches in *catarrhal pneumonia* clear up, whereas in tubercular disease they persist. In cases of *bronchitis* accompaniments are generally heard, either in larger or smaller tubes over the whole of both lungs, but in tubercular disease the accompaniments, generally clicking, moist sounds, are usually limited to the area affected.

Tuberculin has been suggested as a suitable means of testing the presence of tuberculosis, but its use for this purpose has many objections, unless a minute dose of tuberculin T.R. be used for finding the patient's opsonic index (see p. 120).

Prognosis.—There is no more difficult matter to decide than the prognosis of a case of pulmonary tuberculosis. Unquestionably freedom from hereditary taint has an important bearing, and the quiescence of the disease, without any attack of hæmoptysis for at least several years, should make it possible to offer a more favourable prognosis. It should, however, be remembered that patients who have tubercular foci in the lung possess a permanent and irradicable source of danger which should never be left out of account. The danger may be *nil* if the patient exercises prudent care, but there is always the chance of the disease being lighted up to renewed activity,—a chance which should ever be before the mind of the physician, when making suggestions as to the patient's future life and work.

It is wise to endeavour to prevent the marriage of persons suffering from pulmonary tuberculosis, although it is possible that, in the event of complete quiescence, there may be less tendency for children of the marriage becoming tubercular. There is, however, a want of vitality, or of resistance, or it may be a congenitally weak chest, which can truly be described as the heritage of phthisical parents.

Treatment. *Prophylactic Treatment.*—It is of the greatest importance to impress upon patients who are the subjects of pulmonary tuberculosis that they are infectious, and that the chief danger is due to the sputum. There are many useful inventions by which the sputum can be collected and afterwards destroyed, such as by the aid of strong antiseptics or by fire. A portable flask of some kind must be obtained, and the one chosen should have an arrangement by which it can be thoroughly cleansed. In the bottom of the flask there should be a little 10 per cent solution of carbolic acid, and the

patient should be enjoined always to use the flask for purposes of expectoration. Handkerchiefs should never be employed, and if, from the patient's weakness, something of the kind must be resorted to, rags, which can afterwards be burned, should be used. The patient should sleep alone, and certainly, if possible, either in a room by himself, or at any rate in a separate bed from healthy persons. It is a good precaution to enjoin the patient to wash out the mouth with permanganate of potash, or other antiseptic solution, where the sputum is excessive and teeming with bacilli. On no consideration should persons who have a hereditary predisposition to, or have previously suffered from a chest affection, be permitted to nurse or even to live with phthisical patients.

Open air is probably the chief mode of treatment, and where free access of fresh air is permitted, there is less risk of infection to healthy persons. It is a remarkable fact that, in consumption hospitals, few of the attendants become infected with the disease, unless they have a family tendency, or have allowed their state of health to fall much below par.

General Treatment.—The great treatment of the day may be described as the open-air treatment. Where possible the patient should live, eat, and sleep in the open air. Some cases are not able, owing to the condition of their throats or lungs, to stand the cold, but even for them a modified treatment on the same lines can be easily carried out. Where the patient sleeps under cover, the windows must be wide open, and, if possible, the bedroom should have a south exposure. The patient should wear flannel next the skin, and, while a sufficiency of blankets is necessary, any excess of clothing, which might induce sweating, must be discouraged. A hot bottle in winter is a necessity, and there is no reason why the patient should suffer from excessive chilliness. Much may be done, however, to harden the patients by sponging the chest and neck with cold water every morning, summer and winter, and in time they become acclimatised even to the great cold of a high mountain health resort. The effects of the sun are most beneficial, and with plenty of fresh air and bright sunshine, a patient with a swinging temperature and a poor appetite often rapidly and steadily improves, and reaches the stage when life can once more be enjoyed. It is necessary to avoid draughts, and this is accomplished in many sanatoria by having

revolving screens which shelter the patients from the prevailing winds.

Certain cases of phthisis improve to a remarkable extent when taken to a high altitude, and Davos Platz, St. Moritz, Arosa (all about 6000 feet), and many other Alpine health resorts, are famous for the purity and dryness of the atmosphere, and the large amount of sunshine even during the winter months. Many patients suffer considerably from bronchitis, and they do better at a lower altitude, such as may be obtained at one of the winter health resorts above Montreux, for instance, Château d'Oex, Montana, Caux, and many others. Patients who are extremely ill, and patients who suffer from repeated hæmorrhage, should not be sent to a high altitude; the most satisfactory patients are those whose cases are chronic. For persons to whom the rigour of an Alpine winter is unsuitable the warm, dry air of Egypt, and the bright sunshine of Algeria and the Mediterranean, may be commended; and many patients whose lives would be greatly shortened if they remained in our own land, live more comfortably, and for a longer time, especially during the colder months, at one or other of the innumerable Mediterranean resorts. It should, however, be remembered that phthisis is infectious, and that, if a patient is extremely ill, other persons associated with him in hotels and elsewhere may suffer, while the benefit he obtains personally is often slight. Under no circumstances should a dying patient be sent away from home, because even in the most luxurious hotels, and under the best possible conditions, he cannot have the comforts of home.

Diet.—It is an important matter to make the patient eat, and eat well. It is unwise to stuff our patients, and this method of treatment has to a large extent been superseded, although forced feeding is useful in some cases. It consists in forcing the patient to take large quantities of milk, finely pounded meat (sometimes raw), fish, and other nutritious food, so prepared as to be readily digested; and where the patient will not eat, this method has occasionally proved satisfactory. Dyspepsia should be carefully treated; sometimes the use of pepsin or a bitter tonic may help a capricious appetite; and often the combination of fresh air with wholesome, easily digested food enables a patient to put on weight rapidly.

Medicinal Treatment.—Cod-liver oil is one of the most

important and best of remedies. It is often well taken in one big dose in the forenoon, midway between breakfast and lunch, or in small doses after food. Its taste is readily concealed by the use of a pinch of salt, a little bit of dry bread eaten after the dose, or a squeeze of lemon juice; and in the form of emulsions, cod-liver oil, although diluted, frequently acts well. It should never be given where waxy degeneration is present.

General tonics, such as iron, strychnine, arsenic, and most of the mineral acids, are extremely beneficial; but arsenic, which used to be given in enormous doses in the form of the insoluble cacodylate of soda, should be used with considerable caution.

Perhaps the most valued drugs are those which are believed to act as antiseptics. Guaiacol and creosote are admirable remedies, combined with the cod-liver oil, or given alone in capsules, the only trouble being that the stomach is apt to be upset, and that the taste comes back into the mouth many times after the capsule has been swallowed. Guaiacol carbonate, a tasteless powder, given in 20 grain doses thrice daily, sometimes exercises a salutary influence on the patient, but unfortunately its success is by no means certain. Turpentine, in 15-30 minim capsules, is to a large extent excreted by the lung, and has been found serviceable in some cases, while iodoform administered internally as a pill (gr. 3) has been recommended as of distinct advantage.

Special Methods of Treatment and the Treatment of Symptoms.—While the use of tonics and the internal administration of guaiacol and creosote may benefit the lung, a further attempt should be made to treat the local condition. Intratracheal injections of guaiacol 2 parts, menthol 10 parts, in sterilised olive oil 88 parts, may be used, and about a drachm of the mixture injected twice or thrice a day. It is, however, only a chance if much, or any, of the injection reaches the affected part of the lung, and therefore it has been suggested that injections of iodoform or other antiseptics should be given through the chest wall. The objection to this procedure is obvious, that in addition to the conceivable risk of pneumothorax, there is always the possibility of a septic needle track being left when the syringe is withdrawn. Surgical procedure has in certain cases been attempted, but the results are not satisfactory. An extremely useful method of administering

iodoform is to give an emulsion of the drug as an intravenous injection, and the results, in certain chronic cases, have been favourable, while there is little risk of phlebitis being set up at the site of injection. Almost immediately after the injection has been given, the patient's breath smells of iodoform, and coughing is induced.

Inhalations are of great value in attempting local treatment of the lung; they may consist in finely divided particles of a volatile antiseptic inhaled by the patient, and amongst the best agents to use are the essential oil of cinnamon, the essential oil of peppermint, creosote, and guaiacol. Some of these are administered in such a way, that the patient inhales the drug both through the nose and the mouth, and in the case of creosote and guaiacol the remedies are often mixed with parolein or other agents for the purpose of dilution.

The tuberculin methods of treatment are mentioned under the treatment of tubercular disease generally, because while the old tuberculin prepared by Koch is practically never used, the modifications at present under trial appear to have their chief value in the treatment of lupus, tubercular glands, and tuberculosis of an extremely chronic nature, and it is only in cases of very chronic pulmonary tuberculosis that, with our present knowledge, the treatment is worthy of trial (see reference to the action of tuberculin on the opsonic index at p. 120).

Counter-irritation deserves continuous and careful attention. Iodine may be painted on over the affected side of the chest, either the tincture, or a mixture of tincture and liniment, depending on whether the patient's skin is susceptible or not to the drug. Fly-blisters are also valuable, and sometimes the use of a mustard leaf helps to relieve pain and cough.

Cough is often troublesome, and may require sedative cough mixtures. Perhaps one of the best is the Brompton mixture, which consists of dilute hydrocyanic acid (℥ 3), dilute nitric acid (℥ 10), liquor morphinæ (℥ 5-10), syrup of tolu (℥ 15), and acid infusion of roses (ad $\frac{1}{2}$ oz.). Such a remedy is of special value at night, so as to soothe irritation and aid in inducing sleep; and there are endless prescriptions of different kinds which are suitable for the alleviation of cough. It should be remembered that gargling the throat with a little cold water, or drinking a cupful of warm milk or weak tea, frequently checks the most distressing bronchial irritation.

The *pyrexia* requires careful attention, and the remedy almost universally given is quinine, but the necessity for it may be greatly lessened if cold sponging is resorted to, should the temperature rise above 102° F.; and, in fact, the sponging of a phthisical patient at night, not necessarily with water which is absolutely cold, renders him much more comfortable, and frequently prevents the temperature from reaching a high level. On no account should the usual antipyretic group of remedies be employed unless after mature consideration. They tend to increase the amount of sweating, and in many cases probably do more harm than good.

The excessive *sweating* may require special treatment. Probably atropine in doses of $\frac{1}{100}$ th gr. to $\frac{1}{50}$ th gr. is the best remedy in most cases. Quinine is also of value, but it is important to remember that, if the temperature be kept within bounds, the sweating is greatly diminished. Every phthisical patient should be clad in wool during the night, because, even in the mildest cases, there is apt to be greater perspiration than in health.

Should *hæmoptysis* occur, an ice-bag may be placed over the chest, and, if possible, over the bleeding point, while morphia and mineral acids are valuable in soothing nervousness and excitement. Keep the patient absolutely at rest and on low diet. There are many remedies which may also be given, such as turpentine (℥ 10-20), hamamelis tincture (℥ 20-60), ergotin (gr. 2-5). A purge is often of service in lessening the tendency to a recurrence of the bleeding.

Where the intestine becomes secondarily involved, *diarrhœa* frequently results. Its treatment is mentioned under enteritis, but a useful preparation is the lead and opium pill.

The clothing during the day should be warm without being excessive; many consumptives wear an abnormal number of woollen garments, and this should be discouraged, because it is of the greatest importance to harden, rather than to coddle, phthisical patients. Exercise must be graduated according to the patient's strength. It is advantageous to perform pulmonary gymnastics, but the expansion of the lungs is certainly better carried out by gentle exercise than by other means, and if the patient is living in a suitable climate, he should be able to take walking exercise nearly every day, and in bad weather pulmonary gymnastics could be substituted.

The subject of sanatoria is one which has given rise to much discussion; many patients improve greatly in these institutions, because in them they have the advantage of open air and suitable feeding, together with other treatment; but there is little reason why the patient, if living in the country, may not be treated on the same lines at his own home, provided always that home is in good surroundings. On the other hand, the sanatoria situated at high altitudes are of undoubted advantage, and those at Davos Platz, Arosa, and many other places are arranged on specially hygienic principles. Excitement of all kinds is undesirable, and a quiet, well-regulated life should be enjoined, with freedom from overheated rooms and from excessive exercise, such as dancing, and even skating, but each case must be judged and treated on its own merits.

XII. PNEUMONOCOONIOSIS

THE term includes the group of diseases which are the result of the inhalation of dust; the commonest of these are anthracosis (coal-miner's lung), chalicosis (stone-mason's lung), and siderosis (steel- or iron-grinder's lung). *Silicosis*

Etiology.—Dust particles of different kinds cause irritation; they are as a rule taken up by the leucocytes and mucous corpuscles, and, aided by the ciliated epithelial cells, they should be expelled in the mucus by the act of coughing. These particles of dust may reach the air vesicles, and entering the lymphatics pass to the peribronchial lymph glands, or by lymphatic channels running along the interlobular septa they may even reach the pleura, while many of the particles are also found in the lymph glands at the root of the lung. Probably steel, iron, and stone dust are among the most irritating particles, and, where they are present in large quantity in the lymphatic system, they are liable to cause ulceration, but the degree of ulceration depends on the irritating nature of the dust in question. In this way, although coal-dust is by no means a desirable substance in the lungs, it may cause no ulcerative lesion at all; on the other hand, in marked cases of siderosis or chalicosis, ulceration is the rule, and, very frequently, the affected lung becomes secondarily involved by the tubercle bacillus.

Pathological Anatomy.—The process is really a form of bronchitis, with the distribution of the dust particles along the line of the peribronchial lymphatics and the interstitial tissue of the lung. It seems probable that where the mucous membrane of the bronchial tubes remains intact, no serious degree of pneumoconiosis can result. Stone-mason's lung is peculiarly common amongst workers who use covered sheds, or where the kind of stone being cut forms a very fine and irritating dust.

The **Clinical Features** depend greatly on the irritant qualities of the inhaled dust. Cough and gradual loss of health, with the formation of cavities, are the chief clinical features. Frequently particles of the dust in question may be seen under the microscope in the sputum along with elastic tissue, the evidence of disintegration. Hæmorrhage is not infrequent, and a varying amount of dyspnœa develops as the disease progresses.

Diagnosis.—The history of the patient's occupation, or previous occupation, together with a severe bronchitis and the physical signs of cavity formation, should suggest one of the more serious forms of pneumoconiosis. In coal-miner's lung, the so-called black spit is very typical, although there may be little evidence of any ulcerative process.

The **Prognosis** depends on the form of the disease from which the patient is suffering; the more serious forms are not infrequently fatal, although most probably in very many cases death is hastened by the addition of the tubercle bacillus to the pre-existing disease.

The **Treatment** is practically the same as that recommended for chronic bronchitis, and in addition cod-liver oil, iron, and general tonics are of great advantage. The work from which the patient has suffered should be stopped, or means used to prevent the dust being inhaled. In many factories in which dust is almost inevitable, preventive measures are taken to render the dust harmless to the workers. Masks are sometimes worn, and, where possible, the dust is moistened so that it may not become mixed with the air.

It will be readily understood that besides the trades just mentioned, there are many others in which dust may enter the lung; one of these is associated with the dust from grain, in which spores of fungi may not infrequently be found. Appar-

ently the fungus which most commonly causes trouble is the *Aspergillus fumigatus*, and the condition has been called aspergillosis. There are two trades in which aspergillosis may occur, namely, that of workers among human hair who use rice dust, and that of persons who train carrier pigeons, an occupation which is largely carried on in Paris, and the method of which is peculiarly deleterious to those employed. Grains of barley or rice are held between the lips, and the pigeon is taught to take the grain, but, unfortunately, spores of the fungi are not infrequently found adhering to many of the cereals, and as a result the trainer may get the spores introduced into the lung. The spores set up an excessively acute destructive process, closely resembling tubercular disease of the lung, but the prognosis is by no means so unfavourable as in that condition.

XIII. CIRRHOSIS OF THE LUNG

(FIBROID PHTHISIS)

GREAT increase of the fibrous tissue of the lung with consequent invasion of air vesicles. It may be a diffuse condition, or localised to a limited part of the lung previously involved by tuberculosis, pneumonia which has not properly cleared up, or any other disease which has, directly or indirectly, resulted in increased formation of connective tissue.

Etiology.—(1) The most common cause of fibroid lung is certainly chronic tubercular disease. (2) It may in rare cases be secondary to lobar pneumonia, where the affected lung is replaced by fibrous tissue, forming a sequel to that disease which is not particularly common. (3) It may be secondary to chronic bronchitis, with great thickening of the bronchial walls. In this there is gradual involvement of the air vesicles, either in one lobe or possibly in a whole lung, and it is a frequent result of chronic broncho-pneumonia. (4) As a sequel to pneumoconiosis, cirrhosis of the lung may occur. (5) Not infrequently, where there is pleurisy with thickening of the affected pleura, strands of fibrous tissue penetrate the lung. (6) Syphilis of the lung causes a variety of cirrhosis, generally limited to the interstitial septa near the root of the lung.

It should also be added that wherever the lung, or a part

of a lung, is interfered with, either by tumour or other cause, cirrhotic changes occur in the affected area, the amount varying, however, in different cases.

Pathological Anatomy.—Where a whole lung is affected, there is marked flattening of the corresponding side, with nearly always a definite degree of spinal curvature; the pleura is generally greatly thickened, and the air vesicles are encroached upon by the thickened fibrous septa. There is considerable dilatation of the bronchi, forming bronchiectatic cavities of varying size, and in tubercular cases there may be much ulceration associated. Neighbouring organs are drawn over towards the affected side, and generally, if one lung alone is involved, the other shows marked emphysema. Where the cirrhosis is secondary to broncho-pneumonia the areas are scattered, and may be separated by emphysematous lung. Occasionally small aneurisms are present on branches of the pulmonary artery in one or more of the cavities, and if there be much pus formation, waxy degeneration is present in the liver, spleen, kidney, intestine, and possibly other organs.

Clinical Features.—There is always a prolonged history leading up to the cirrhotic changes in the lung, which are very frequently of tubercular nature. In these cases the clinical features are those of chronic tuberculosis, in others there may be more thickening of the pleura, while bronchiectatic cavities give rise to the usual physical signs associated with them. There may be copious and purulent expectoration, and often great dyspnoea, but the degree depends largely on the pathological changes in the lung and the extent of its involvement.

Physical Signs.—*Inspection.*—The immobility of the affected lung, and its marked retraction, are extremely typical. Generally the opposite lung is emphysematous, and frequently, as a result of the disease, there is spinal curvature. *Palpation* may show diminution or loss of vocal fremitus, or sometimes increase, while on *Percussion* a dull note is obtained, with here and there evidence of the existence of cavities; a definite tympanitic note is often much interfered with by the greatly thickened pleura. On *Auscultation*, the breath murmurs are generally distant bronchial, or cavernous, the vocal resonance is usually increased, and there may be whispering pectoriloquy, while the quantity of the moist sounds depends on the amount.

of secretion present in the part of the lung under examination.

Displacement of neighbouring organs is always a marked feature, especially where the whole of one lung is fibroid. A fatal result is not infrequently due to exhaustion, sometimes to waxy degeneration, and occasionally to hæmorrhage.

The **Diagnosis** is rarely difficult, although it is important to make out a tubercular element in the case, and, generally speaking, the recognition of tubercle bacilli in the sputum is easy.

The **Prognosis** depends much on the degree of the condition and on its nature. If primarily tubercular, or if tubercle has secondarily involved the lung, the prognosis is more grave, but much depends on the ability of the lung to carry on the work of life.

The **Treatment** is largely directed to the relief of cough and to an attempt to produce drying up of the secretion, while if the sputum has become foetid, every effort must be made to render it aseptic. It is important to remember, where a patient is sufficiently wealthy to choose his own climate, that a warm, dry health resort, in winter, is beneficial, but that no amount of treatment, medicinal or climatic, can actually cure, in the sense of removing altogether, the condition from which the patient is suffering.

XIV. PULMONARY EMBOLISM AND THROMBOSIS

PULMONARY EMBOLISM

WHERE a clot or embolus enters one of the branches of the pulmonary artery, it causes pulmonary embolism.

Etiology.—It may be due to phlebitis of any systemic vein and the entrance of a clot into the venous circulation. Such a clot may also originate from one of the uterine veins in puerperal septicæmia. In valvular lesions of the heart, and especially in mitral stenosis, an embolus may be separated from a clot in the right auricle or ventricle, or from vegetations at the tricuspid or pulmonary orifices. In fracture of the long bones, fat embolism may occur in the lung.

Pathological Anatomy.—A hæmorrhagic infarct occurs, blood pouring into the affected area from surrounding vessels,

and it may contain suppurative organisms, in which case the area of infarction breaks down and forms an abscess. Limited pleurisy is generally present, and in cases where the infarct breaks down, the abscess may open into the pleural sac.

The **Clinical Features** depend largely on whether the infarction does, or does not, contain suppurative organisms. Often a definite area of dulness can be made out, over which the breathing is bronchial. Hæmoptysis lasting for several days is generally present, and pleuritic pain is common. Dyspnoea may be severe if much lung is involved, and there may be symptoms indicating the breaking down of septic infarcts.

The **Diagnosis** is usually aided by the hæmoptysis, the pleuritic pain, and the limited area or areas of dulness.

The **Prognosis** depends on the nature of the infarction. A simple infarction may actually relieve a heart case, where backward pressure is extreme and the consequent hæmoptysis is free. In septic cases the outlook is grave in the extreme.

The **Treatment** must be palliative.

PULMONARY THROMBOSIS

This sometimes occurs in croupous pneumonia, pulmonary tuberculosis, tumours, and other pulmonary lesions, and is always of grave import. Gangrene may ensue, and rapidly kill the patient.

XV. ABSCESS OF THE LUNG

Etiology.—(1) In pulmonary consolidation, whether it be lobar pneumonia, lobular pneumonia, pulmonary infarction, or tuberculosis, the lung may, as the result of infection with suppurative organisms, and partly perhaps on account of diminution in the vitality of the tissue, break down and form an abscess. In the same group we may include abscess associated with cancer or other new growths, although it should be stated that tumour more probably causes abscess formation as a secondary result by septic pneumonia, than from suppurative change occurring in connection with the tumour masses themselves.

(2) Pyæmia causes multiple abscess formation, either in the line of the pulmonary artery, where an ulcerative endocarditis

specially involves the right side of the heart, or by the terminal branches of the general arteries which supply the bronchi, in which case the suppurative organisms are distributed from the left side of the heart. It should, however, be stated that as organisms, capable of causing suppurative foci, may pass through capillaries, in an ulcerative endocarditis of the left side of the heart, the organisms may readily reach the lung. The focus of distribution may be in other positions besides the cardiac valves; for example, it may be found in the septic clot from a phlebitis or in the uterine wall in puerperal septicæmia.

(3) Pulmonary abscess may also arise from perforation of a neighbouring abscess into the lung. This has occurred in cases of tropical or other abscess of the liver.

Pathological Anatomy.—The abscesses noted above differ greatly in size, position, and clinical features, and they may in certain cases be associated sooner or later with gangrene.

The **Clinical Features** vary greatly. There may be marked evidences of septic absorption, with a swinging temperature and much sweating, while in other less acute cases the patient suffers comparatively slightly. Not infrequently the abscess discharges by the bronchus or opens into the pleura.

The Physical Signs may resemble those of a cavity, associated often with the expectoration of a large quantity of purulent material.

The **Diagnosis** is by no means always easy. The tendency to fætor of the breath and the stinking character of the expectoration, with evident breaking down of lung, should make the physician suspect the presence of an abscess, although the history of each case is of great importance. Sometimes the development of pleurisy reveals the fact that the abscess is in close proximity to the pleura.

The **Prognosis** in many cases is grave, and in pyæmia death is the only likely termination. There is also the risk of the abscess opening into the pleura and so inducing pyothorax.

The **Treatment** is mainly surgical, although one hesitates to recommend surgical operations which involve the lung itself. It is, however, the only method likely to result in benefit to the patient, and the most recent results are encouraging. Quinine is, perhaps, the one drug which should be given, owing to its effect in counteracting septic absorption.

XVI. GANGRENE OF THE LUNG

GANGRENOUS destruction of one or more portions of lung.

Etiology.—Gangrene depends on the presence of certain putrefactive organisms, associated with a lowered tissue vitality which permits of their growth.

There are three groups of causes :—

1. It may be due, or secondary, to the inspiration of food, infective blood, septic discharges from nose, mouth, or larynx, and possibly to the entrance of a foreign body.

2. It may be secondary to some pre-existing lung disease, and especially bronchiectasis, lobar pneumonia, septic pneumonia as from pressure of a tumour on trachea or bronchus, and, lastly, tubercular disease of lung. In croupous pneumonia and pulmonary phthisis, excessive debility and the devitalising influences of chronic alcoholism are important factors.

Thrombosis of a large branch of the pulmonary artery is often associated with the diseases mentioned above, and may be the direct cause of the gangrene.

3. It may spread from neighbouring septic foci situated in or near the œsophagus, stomach, mediastinum, or pleura, and metastatic inflammation, resulting in gangrene, may occur as in pyæmia, the original lesion being a septic uterus, bedsores, etc. Usually in this latter group a septic embolus is the vehicle by which the organisms are conveyed to the lung.

Pathological Anatomy.—The lung involved is greenish-brown or blackish, with most offensive smell, and generally there is breaking-down in the centre. It may be limited, or involve a whole lung. The gangrenous area is usually surrounded by a zone of extremely congested, often pneumonic, lung.

Clinical Features.—There is marked depression of vital powers, malaise, and frequently diarrhœa from septic absorption. It may be insidious in onset. Soon the appalling odour of breath and sputum aids in the diagnosis. The sputum separates into three layers. At the bottom of the vessel it is greenish-yellow or brown, and more solid, and contains much *débris* of lung (elastic tissue), fatty acids, micro-organisms, etc. The middle layer is opalescent, and on the top the sputum is covered with brownish froth. Hæmoptysis sometimes occurs.

OF MEDICINE

and the patient's strength is

perhaps the evidence of
constant, and moist râles occur in
signs of the pre-existing dis-

amply clear, owing to the factor
tissue in the sputum.

generally death from exhaustion
sometimes with the development of a
consequent and very intense dyspnoea.

in cases in which the gangrene is
when a local gangrenous abscess may
externally or internally into a bronchus.

patient's strength must be kept up by
means.

Vaporise antiseptics in the

patients and attendants, using car-

or similar agents. Try intratracheal

parts, menthol 1.0 parts, and sterilised

daily). (Give quinine, terebene,

may counteract the toxins, and possibly,

may exert a local antiseptic influence,

the antiseptic is introduced direct

the chest wall. Surgical interference

but should always be considered.

which may arise.

NEW GROWTHS OF THE LUNG

generally met with in the form of lympho-

in connection with the glands at the root

a tumour will cause many pressure pheno-

pleural effusion: death is generally due

while it may involve the lungs along

shows no very characteristic clinical features.

The tumour may be cartilaginous in nature, but vary con-

siderably, according to the type of the primary tumour. Rare.

of the lung is rare: it not infrequently

develops in the neighbourhood of the root of the lung, and

spreads along the bronchi, in time infiltrating the greater part of one lung. Tumours of this kind cause marked stridor from stricture of the bronchial tubes, and very frequently, owing to ulceration, there is a considerable amount of hæmorrhage, the bleeding being rather continuous than excessive; the sputum is described as red-currant-jelly or sometimes prune-juice-like in character.

Secondary cancer of the lung is a not uncommon result of a primary tumour of the breast, the stomach, or some other organ. These tumours are often encephaloid, sometimes melanotic cancers (where the eye or skin is the site of the primary growth); usually there are numerous nodules varying greatly in size, some of them large, being about as big as a small orange, and others small, almost resembling miliary tubercles. They may involve the pleura and cause considerable pleural effusions; such pleural effusions are often hæmorrhagic.

The **Clinical Features** have been so far referred to, and, as already indicated, they vary greatly, according to the site and nature of the different growths, but pain, cough, and some definite change in the breath sounds, and often evidence of pleural effusion, may be said to be typical of most cases of malignant disease of the lungs.

The *Physical Signs* also vary considerably; there may be evidence of secondary growths in connection with the ribs, or there may be dilated veins indicating pressure on the superior vena cava or some of its branches. The percussion note over the region of the affected lung is usually dull, and the stridor in the breath murmurs is very typical where there is partial occlusion of a bronchus.

The degree of general disturbance depends on the nature and distribution of the tumours. Emaciation may be rapid, or the patient may live in moderate comfort for a period of months.

Diagnosis.—Where the primary tumour, whether of the breast, stomach, or other organ, has been diagnosed, the involvement of the lung or pleura is easily recognised. There is no question that the presence of blood in the sputum, so common in primary cases of cancer, should render it imperative to overhaul the lungs with special care, and the investigation of the percussion dulness and breath murmurs at the

roots of the lungs may help when difficulty arises. Sometimes the cervical or other lymphatic glands show evidence of secondary growths.

Prognosis.—Death most frequently results from pressure, sometimes from emaciation, and certainly septic pneumonia is the final stage in many cases where the lung condition is mainly responsible for the death of the patient.

The **Treatment** must be purely palliative; the use of opium and attention to the tapping of the pleural sac, when there is much effusion, are the most important indications.

XVIII. SYPHILIS OF THE LUNG

SYPHILIS of the lung has been mentioned already in connection with syphilis generally; it is therefore only necessary to remind the reader that in congenital cases syphilitic cirrhosis of the lung—so-called white pneumonia or white hepatisation—may occur, and that in tertiary syphilis gummata are not infrequent near the root of the lung. Further, stenosis of the trachea or bronchial tubes may result from syphilitic ulceration generally with gummata. The clinical features are not distinctive; the white pneumonia of the infant is often incompatible with life, and the presence of gummata may rather be guessed at than diagnosed by any special clinical feature. The treatment should be on antispecific lines.

XIX. HYDATID CYST OF THE LUNG

It is not common to find hydatid cysts in the lung, the liver and kidneys being relatively much more frequently affected. The cyst may be in the lung or pleura, and may grow to a considerable size. The symptoms are cough, pain in the chest, a varying amount of dyspnoea and sometimes hæmoptysis; while there may be definite evidences of the cyst such as local bulging of the chest wall and a dull note on percussion. Sometimes neighbouring organs are displaced.

The **Diagnosis** may be difficult, but is often cleared up after an exploratory puncture is made, the fluid being generally

typical in its appearance, and it sometimes contains hydatid hooklets.

The **Prognosis** is always grave, because other cysts may be present, and suppuration and possibly gangrene may ensue.

The **Treatment** should certainly consist in the tapping of the cyst in the hope that it may not fill up again, but sometimes a free incision is called for.

XX. DISEASES OF THE PLEURA

(1) ACUTE PLEURISY

INFLAMMATION of the pleura, which may be arrested at what has been called the *first* stage of pleurisy—the stage when fibrinous lymph is alone exuded. In many cases a further, or *second*, stage is reached in which a more serous effusion follows the fibrinous exudation. The *third* stage consists in the removal by absorption of the serous lymph, and practically corresponds in symptoms and signs to the first stage.

Etiology.—In a large number of cases pleurisy is of tubercular origin. At one time too little importance was attached to this association of tubercle and pleurisy, and there seems now to be a greater risk of associating too many cases of obviously non-tubercular pleurisy with the tubercle bacillus. Pleurisy is a common result of exposure to cold or wet, and as such it may occur in a perfectly healthy individual, although it is more likely to develop in one with a tubercular tendency; it is possible that some of these cases ascribed to cold may be of rheumatic origin.

Pleurisy is also secondary to the following diseases—pneumonia, cancer, abscess, gangrene, and infarction. Not infrequently a pleurisy, which is rather more subacute than chronic, is associated with the terminal stages of Bright's disease, cirrhosis of the liver, and cancer other than that directly involving lung or pleura,—a group of causes, therefore, which may sometimes be toxic in nature, although in most cases the pleurisy is due to infection by an organism.

From what has already been said, it may be gathered that in many pleural effusions definite organisms are present to which the pleurisy is directly due; these include the Tubercle

bacillus, Pneumococcus of Fränkel, the Diplobacillus of Friedländer, Streptococci and Staphylococci, and sometimes the Diphtheria bacillus, the Typhoid bacillus, the Bacillus coli, the Bacillus aerogenes capsulatus, and, less frequently, many other organisms.

In any or all of these, an effusion may occur of more serous lymph.

Pathological Anatomy.—There is first congestion of the pleural surfaces, followed by the exudation of fibrinous lymph varying much in amount in different cases. The serous effusion is poured out later; it is pale yellow in colour, and may contain more or less flocculent material, and in some cases a considerable amount of blood. On microscopic examination, in addition to numerous endothelial cells there are also leucocytes, and much significance has recently been attached to the variety of leucocytes found in the fluid. Where the majority of the cells are lymphocytes, the pleurisy is believed to be closely associated with a tubercular cause, but in order to carry out the test it may be necessary to centrifuge some of the effusion so as to obtain the majority of the cells present. There is always a comparatively large amount of albumin, much larger than what is found in dropsical effusions. In certain cases the fluid forms a jelly outside the body, and this is common in cases in which it closely resembles in constituent parts the serum of the blood. Bile, sugar, and sometimes uric acid and cholesterin may be found in the fluid, and when a suppurative organism is present, pus cells are seen under the microscope in enormous numbers. The quantity of serous lymph varies from 60 to 100 oz., or even more. Where there is much effusion, the lung is compressed, and is pushed upwards and backwards unless limited by previous adhesions. Should effusions remain long in position, layers of organised lymph may form on the visceral pleura, and greatly restrict possible re-expansion, and for this reason it is probably unwise to permit a large effusion to remain in the pleural sac longer than two or three weeks.

Where there are extensive effusions, there is always much displacement of neighbouring organs, and the heart may be pushed over in an effusion of the left side, so that the apex-beat is discovered to be to the right of the sternum. Such great displacements of heart and vessels not merely cause

palpitation and discomfort, but might actually lead to a fatal result, although fortunately this is rare.

When the effusion has become absorbed, the fibrinous lymph on the visceral and costal pleuræ may either completely disappear, may organise to a certain extent, or may form adhesions greatly interfering with the action of the affected lung. In these adhesions calcareous deposits sometimes occur, but more commonly great thickening of the pleura results, and an invasion of the neighbouring lung by the connective tissue is also seen along the lines of the interlobular septa.

Clinical Features.—There is generally a chill or rigor with severe pain in the side, although in a small proportion of cases a pleurisy may develop insidiously and without pain. Pleuritic pain is sharp, is often described as stabbing in character, is accompanied by cough, and is much increased on deep inspiration. As a rule it is localised, and its position suggests the site of the pleurisy; in exceptional cases the pain is referred to some distant point, and such referred pain is perhaps specially common in connection with diaphragmatic pleurisy. The temperature is generally found to be high, usually 102° or 103° F., but during the stage of serous effusion it may drop to normal.

The *Physical Signs* of this the *first* stage of pleurisy are usually simple and diagnostic. On *Inspection*, the efforts to limit respiratory movement on the affected side are very obvious, and the breathing is generally shallow and rapid. On *Palpation*, in certain cases friction fremitus may be made out. *Percussion* rarely yields any obvious increase of dulness during this early stage, and on *Auscultation* the breath murmurs are indistinct, rather from the patient's efforts at limiting movement than from the fibrinous lymph interfering with the conduction of sounds. The chief auscultatory sign is friction, heard most distinctly at the end of inspiration and the beginning of expiration, greatly increased in distinctness by the patient taking a deep breath, and not altered in character by coughing. These features prevent confusion between friction and moist râles. Pressure with the stethoscope generally increases the friction sound. It should not be forgotten, however, that interlobar pleurisy and diaphragmatic pleurisy may, at all events to begin with, and sometimes throughout their whole course, give rise to no audible friction owing to the

distance of the affected surfaces of pleura from the chest wall.

A pleurisy may never pass beyond this first stage, and the application of a blister or other simple treatment often completes the cure in a few days.

Sero-fibrinous Pleurisy. — This corresponds to the *second* stage of a pleurisy with effusion, and after the physical signs of a dry pleurisy have been more or less definitely made out, effusion occurs. It is usual to find that the pouring out of a serous effusion causes the disappearance of the pleuritic pain, because it separates the two layers of pleuræ from each other, although friction is often still audible at the upper limits of the effusion.

The patient generally lies in bed either on his back or on the affected side. Not infrequently there is a considerable amount of dyspnœa.

Physical Signs.—On *Inspection*, where the effusion is considerable in amount, the affected side is seen to be immobile. It bulges and the lower interspaces are pushed outwards, and with the cyrtometer it is found that in extensive effusions the measurement of the affected side greatly exceeds that of the healthy side. In the epigastric region we may note diminished movement of the diaphragm, and with it of the abdominal wall on the affected side. On *Palpation*, we recognise the diminished movement, and also the absence of vocal fremitus. *Percussion* yields a note which is frequently described as wooden, although it is really more bone-like in character. Fluid is excessively resistant, and, with the hammer finger, it is easy to recognise the enormous increase of this resistance. Above the effusion, where the lung is pushed upwards, a tympanitic note is obtained, due to the air in the lung vibrating as one column; the phenomenon depends on the relaxation of lung tissue. The upper limit of the effusion varies somewhat at different points on the chest. It is generally highest in the axillary region, and is at a somewhat lower level close to the vertebral column and at a still lower level at the sternum, but a good deal depends on the amount of the effusion, and also on the position of the patient, because fluid in the pleura follows, to a certain extent, the laws of gravity. *Auscultation.*—While a friction rub may still be audible, and often is audible, at the upper limit of effusions, the breath murmurs are found to be absent

Grouse's sign.

altogether over the chest. Below the level of effusion is very ex- scapular region may is also lost, although the diminution be- limit a peculiar re- recognised. It is as described by Laen- from which resemb- believed to be due- the voice sounds pas-

The physical sig- liver, should be care- doubt with regard to- dermic syringe, and- tion has the advanta- presence of fluid cert-

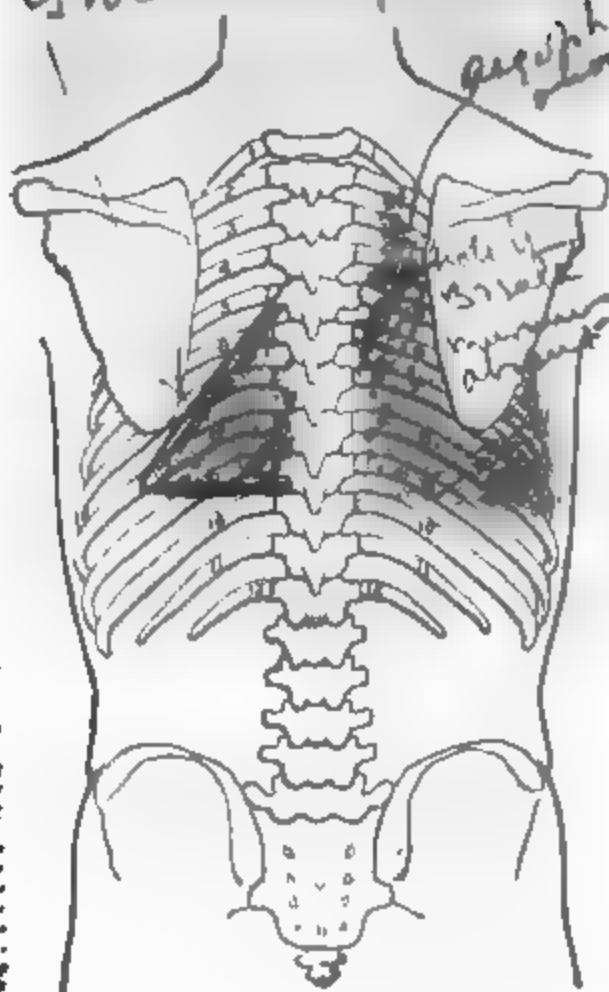
cell constituents, and - - - - - whether the fluid is prob-

ably of tubercular origin or not, and if it is becoming purulent. As a general rule the fluid will become absorbed in one to two weeks, the level gradually falling, and as the layers of the pleuræ come in contact once more, friction, very often excessively coarse, is audible and generally palpable. This has been termed *redux-friction*, and in a favourable case complete disappearance of the effusion may eventually result. In children, certainly, this is by no means uncommon, whereas in adults it is perhaps more likely that some evidence of the effusion will persist throughout life. A leucocytosis in the blood usually develops in the course of an effusion, and albumoses may appear in the urine during the stage of absorption.

Diaphragmatic Pleurisy is only peculiar inasmuch as initial pain is frequently referred to the corresponding shoulder, the abdomen is kept extremely rigid, and an effusion may take a considerable time before percussion reveals a definite area of dulness.

A *Latent Pleurisy* has been already mentioned; there is usually the history of a chill, but no pain, and the patient may seek relief on account of the dyspnoea from which he suffers upon any exertion.

An *Interlobar Pleurisy* and an *Encysted Pleurisy* may



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present considerable difficulty. *Interlobar pleurisy* is not infrequently secondary to pulmonary infarction, and pleuritic friction may only be heard some little time after pain and cough have made us suspect the presence of the pleurisy. An *Encysted Pleurisy* implies the existence of adhesions, and it may be necessary to use the needle before the nature of the case is cleared up.

An *Empyema* is not infrequently at first a sero-fibrinous pleurisy, and the reason why the fluid becomes purulent is due to its infection by suppurative organisms. The clinical features of empyema are described separately.

Diagnosis.—The physical signs of an effusion are sufficiently diagnostic, although where the pleura is greatly thickened, there may be some difficulty. When it is remembered, however, that a hypodermic needle can be used as a final and positive test, the diagnosis at once becomes clear. It is extremely unlikely that a *pneumonic* or *tubercular* consolidation can be mistaken for a pleural effusion, and it is only in the rarest possible cases that bronchial breathing and increased, in place of diminished or absent, vocal resonance are associated with pleural effusions. Where a *bronchial tube* is *blocked*, although the breath sounds are absent and the vocal resonance greatly diminished, it is unlikely that the percussion note will be so dull or the resistance so great as over a pleural effusion. The cause of the effusion is often elucidated by an investigation of the cells, by the amount of albumin, and perhaps by the presence of blood. A *hydatid cyst* in the lung may closely resemble a pleural effusion, and a similar difficulty may be associated with a hydatid cyst in the right lobe of the liver. Exploratory puncture should, however, yield definite data from which a diagnosis can be made.

Prognosis.—Generally speaking the prognosis is good; the only difficulty is the possible tubercular element in the case, and any other causal factor the significance of which may in itself be grave. Thus the pleural effusions associated with cancer are invariably of grave import.

Treatment.—For the first stage of pleurisy, or for cases of dry pleurisy, counter-irritation is probably the best possible mode of treatment. The application of one or two small fly-blisters, perhaps 1 in. × 2 ins., or painting on liquor epispasticus or strong iodine, and the simple but less

efficacious mustard leaf, are excellent modes of combating the condition. Where there is much pain and irritating cough, fomentations or poultices may be preferred. Strapping the side with plaster so as to limit movement, and the application of a bandage, are often useful where pain and cough are very severe. The ice-bag has been much commended, but it is probably more applicable in pneumonic pleurisies than in those due to other causes.

As in every case of inflammation, a purgative forms a useful part of the treatment; perhaps a dose of salts is more suitable than anything else, while diuretics and, where necessary, diaphoretics may also be given. Should the cough be unusually troublesome, powerful sedatives are called for, and even the use of morphia should be entertained, if the cough prevents sleep. It is wise also to study any constitutional element which may be present, and hence the salicylic acid group should be given in cases with a rheumatic history.

Where an effusion is present, it is important to endeavour to secure its absorption as rapidly as possible, and this should be done without tapping in the majority of cases. Purgatives, diuretics, diaphoretics, and limiting to a certain extent the amount of fluid drunk by the patient, will all be found of value. On the other hand, it should be remembered that an effusion must on no consideration be allowed to remain in the chest longer than two or three weeks, unless by the end of that time absorption has commenced. It is not necessary to remove the whole or even a large amount of the effusion in order to induce more rapid absorption. If there is the slightest suspicion of the fluid becoming purulent, the tapping should be done promptly and thoroughly, although it will probably prove to be insufficient.

Paracentesis.—Tapping should be performed in the 7th or 8th interspace, and either in the posterior axillary line, or better, slightly nearer the spinal column. Care should be taken that the needle puncture is below the level of the effusion, and an attempt may be made to widen the interspaces by making the patient stretch over the arm on the side to be tapped, towards the opposite shoulder, just when about to introduce the needle, tell the patient to take a deep breath. Potain's aspirator is probably the most suitable instrument; the needle and cannula should be thoroughly sterilised, while

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the skin at the site of the puncture should be washed first with soap and water, and thereafter with absolute alcohol, or should have a carbolic fomentation placed over it for some hours before the operation is performed.

Holding the needle in the right hand, the operator should place the thumb of the left hand on the upper margin of the lower rib of the interspace selected, and then, before making the puncture, should pull upon the skin so as to make the opening valvular. The needle should be thrust boldly forwards until it passes into the fluid, when all resistance suddenly ceases. The fluid should be slowly aspirated into the bottle connected with the instrument, care being taken not to allow the end of the cannula to irritate the lung by rubbing against it, and thereby setting up troublesome cough. It is probably unwise to remove more than 50-70 oz. from one pleural sac, inasmuch as the removal of fluid permits of considerable cardiac excitability and over-action; it is therefore prudent in every case, where a considerable amount of fluid has to be tapped, to bandage the chest *during* the operation, the bandage being gradually tightened while the fluid is being withdrawn.

The dangers connected with paracentesis are slight if the operation is carefully performed. Pneumothorax and subcutaneous emphysema are extremely rare, and generally are due to carelessness, while it is very unusual to find that even a puncture of the lung causes much escape of air into the pleural sac. Severe coughing of a paroxysmal nature is due either to the two pleural surfaces rubbing against each other, or to the cannula scraping against the visceral pleura; such coughing is only dangerous when it induces severe hæmorrhage. Syncope is by no means uncommon, and should be guarded against by administering a small dose of alcohol to the patient before, sometimes during, and always after the operation. Ether, in a hypodermic syringe, should also invariably be kept in reserve in case of need. Epileptic seizures have been described, but are very rare. Albuminous expectoration has been recorded as a sequel to paracentesis. It really constitutes a form of œdema of the lungs, causing intense dyspnœa, and may prove fatal; fortunately it is of rare occurrence.

After tapping, renewed and vigorous attempts at counter-

irritation should be carried out, and much benefit will result from persistent painting with iodine over the affected side. Pulmonary gymnastics are certainly of great value; the patient may be made to blow water out of a bottle through a spray, or he should simply take a specified number of deep breaths several times a day.

(2) EMPYEMA; PYOTHORAX

Pus in the pleural cavity.

Etiology.—(1) It may result from a simple pleurisy with effusion, probably as the result of infection by a pus-producing organism. In many so-called sterile empyemas the cause of the condition is pulmonary tuberculosis, although few tubercle bacilli may be discoverable in the pus. (2) Empyema is a common complication of certain fevers, especially in children; pleural effusions in scarlet fever, measles, and some other continued fevers are apt to be purulent. (3) Although pneumonia is a continued fever, it is better to keep its association with empyema distinct from the preceding class, because it may occur in adults as well as in children, and much more frequently. Probably a large number of cases of empyema are really associated with an unrecognised pneumonia. (4) Empyema is also not infrequently the result of infection either from without or within; from without:—a fractured rib or an incised wound, malignant disease of the œsophagus, stomach, or some other organ communicating by ulceration with the pleural sac; from within the lung:—malignant tumours, abscesses, tubercular or pyæmic, and necrotic infarcts may give rise to empyema.

The bacteria associated with empyema are the following:—so-called sterile empyemas are probably tubercular, and in many cases the Pneumococcus of Fränkel is found in pure culture, while Streptococci, Staphylococci, and other less common pus-producing organisms may be met with.

Pathological Anatomy.—The purulent discharge has a peculiar greenish-yellow colour with a heavy, sweetish odour unless in cases where, from infection by putrefactive organisms, it becomes foetid or stinking. There are erosions on the affected surfaces of the pleura, and there may be evidences, post mortem, of the different ways in which pus seeks exit, if

an operation has not been performed. There is marked collapse of the affected lung, which may be bound down by adhesions rendering any great degree of re-expansion extremely improbable.

Clinical Features.—The phenomena described under pleurisy with effusion are closely simulated where the effusion is purulent, but the following differences are worthy of note :—

There is frequently the evidence of pus formation which the swinging temperature chart suggests, and sweating is common. The leucocytes in the blood are almost invariably greatly increased, and there is apt to be more malaise, although not necessarily any greater amount of coughing and pain than with a non-purulent effusion.

Physical Signs.—The skin over the affected half of the chest is sometimes œdematous, and the degree of bulging of the interspaces may be very marked. In children, tubular breathing is occasionally heard over an extensive purulent effusion, but the percussion note is just as dull and bone-like as in a more serous effusion. The dislocation of the heart and mediastinum, and of the liver, where the effusion is on the right side, is more marked in cases of empyema. Rarely does an empyema pulsate in consequence of the communication of the cardiac contractions to it, but a pulsating empyema is more common than a pulsating effusion which is non-purulent.

An empyema may undergo the following changes :—

1. Absorption of the fluid may occur, when extensive *calcareous* deposition generally results.

2. It may perforate in the following ways :—

(a) Very frequently the pus burrows outwards, either in one of the interspaces, frequently about the 5th or 6th, or it has even been known to make its way along the *psoas* muscle into the iliac fossa, simulating a *psoas* abscess. Such external perforation has been called *empyema necessitatis*.

(b) An empyema may either perforate into the lung by one or two considerable openings, or it may soak into the lung as the result of necrotic changes in the pulmonary pleura.

(c) An empyema may open into the stomach, peritoneum, or pericardial sac, and may sometimes cause disastrous results.

Diagnosis.—The hectic temperature, a great increase of leucocytes, more than we should expect in a simple serous effusion, and greater displacement of neighbouring organs, suggest the probability of an empyema, while its relative frequency in children who have scarlet fever or measles is also an important point. The removal of a little of the fluid with an exploring needle clears up any difficulty.

Prognosis.—An empyema generally results in more or less sinking in of the chest. It is decidedly a more grave condition than a simple pleurisy with effusion, and if it is untreated, not merely does it produce permanent collapse of the greater part or the whole of one lung, but the burrowing of the pus, either internally or externally, may cause serious mischief.

Treatment.—It is a difficult matter to urge resection of ribs as the only method of treatment, for in empyema in children aspiration will, in a small number of cases, be found sufficient to effect a cure. In the great majority of instances, however, resection is necessary, and should not be delayed. Many efforts have been made to cause re-expansion of the affected lung by the use of valvular tubes; and sometimes, by the use of a head of water fixed above the patient, and connected by a T-tube both with the patient's chest and with a bottle placed on the floor, an attempt has been made to aid re-expansion, and, further, to make the patient by deep inspirations help the passage of the water from one bottle to the other. The necessity for the irrigation of the cavity with antiseptics depends upon whether the fluid is foetid or not. One extremely important point is the risk of death during the administration of the anæsthetic in empyema; as a rule, it is the result of carelessness in turning the patient over on to his healthy side, thereby interfering with the free entrance of air into the only serviceable lung.

During the later stages of recovery after operation, pulmonary gymnastics are of very great value.

(3) CHRONIC PLEURISY

Many cases of chronic pleurisy result from the more acute affection; sometimes they are associated with a considerable amount of effusion; in other cases there is no effusion, but firm pleuritic adhesions develop, and may eventually cause great

thickening of the pleura. This latter condition is one mode of origin of cirrhosis of the lung.

It is unnecessary to refer specially to chronic pleural effusions, and the phenomena associated with a thickened pleura are generally those present in fibroid or cirrhotic lung. The flattening of the chest on the affected side, the diminished vocal fremitus, the presence of a dull note, almost suggestive of effusion of fluid, the absence of breath murmurs, and the absence or diminution of the vocal resonance, are the chief physical signs of thickened pleura. It should also be remembered that thickened pleura with cirrhotic lung causes traction on neighbouring organs towards the affected side.

Occasionally in cases of chronic pleurisy affecting the region of the right apex, the subclavian artery may be compressed by the adhesions, and a pulsus paradoxus develops in the right radial artery, while sometimes vasomotor phenomena, such as the flushing of one cheek, local sweating, or dilatation of the pupil, may indicate involvement of the cervical or dorsal sympathetic.

(4) HYDROTHORAX

Dropsical effusion into the pleural sac.

Etiology.—It is a result of backward pressure, whether of cardiac or pulmonary origin, while local pressure on vessels may also cause the condition. Obstruction of the portal circulation may bring about hydrothorax as well as ascites, and pressure on the thoracic duct sometimes gives rise to a chylous effusion. In Bright's disease, in malignant disease of the lung or pleura, and in blood diseases, especially of the cachectic type, dropsical effusions may develop.

Pathological Anatomy.—The fluid transudes; there is sometimes slight thickening of the pleuræ, but not to any marked extent, unless the effusion has been of long duration. The fluid contains much less albumin than an inflammatory effusion does; it is straw-coloured, unless it contains much chyle, when it becomes milky, and it varies greatly in amount from 2 or 3 to over 100 ounces. It is usually to be found at the lower parts of the pleural sac, if pleuritic adhesions are not present.

Clinical Features.—In cases of backward pressure of cardiac or pulmonary origin, and in cases of Bright's disease, there is generally very evident dyspnoea, rendered much more

marked by the presence of an extensive hydrothorax, and the dyspnoea is dependent on the amount of the effusion. Pyrexia, if present, is not the direct result of a hydrothorax.

The *Physical Signs* resemble those of pleurisy with effusion, except that the displacement of neighbouring organs is generally not so great, and it is said that the upper limit of the effusion forms a straight line in place of the curve, higher in the axillary region, which was described as being typical of an inflammatory pleural effusion. Too much importance must not, however, be attached to these points.

The **Diagnosis** depends partly on the recognition of the cause of the condition, and partly on the examination of the fluid. Evidence of dropsy elsewhere, both in the lung itself and in the dependent parts of the body, is usually noted.

The **Prognosis** is often grave, because dropsical conditions frequently point to a causal factor, the treatment of which presents great difficulties, and in too many cases the benefit obtained is apt to be purely temporary.

The **Treatment** should be directed towards the removal of the causal condition, and the usual treatment for dropsy generally should be carefully carried out. This includes the use of purgatives, diuretics, diaphoretics in certain cases, and sometimes limiting the consumption of fluids by the patient. The removal of part of the effusion should be attempted in most cases by paracentesis. Occasionally venesection is indicated, but only in cases in which the right heart is tremendously engorged.

There is no special treatment for chylous effusions; in many cases it implies the presence of malignant disease.

See p. 479, note on p. 479.

(5) HÆMOTHORAX

Hæmorrhage into the pleural sac.

Sometimes an inflammatory pleural effusion contains an excessive amount of blood, and this may occur in patients who are the subjects of hæmophilia, or rarely in tubercular disease. It is more commonly the result of trauma, owing to the lung being torn by a fractured rib, or of aneurism, where rupture has occurred into the pleural sac. It is an occasional and somewhat serious sequel to paracentesis, where a large blood-vessel has been punctured.

The clinical features are those of pleural effusion, and the prognosis depends greatly on the nature of the case. The treatment is sometimes directed to an attempt at arresting the hæmorrhage, and for this purpose ergotin and the free use of ice are often of considerable help. Absolute rest should be enjoined, and in traumatic cases resection of the ribs and clearing out the clots have been followed by excellent results.

(6) PNEUMOTHORAX AND PYOPNEUMOTHORAX

Air or gas in the pleural sac, in many cases associated also with the presence of fluid often purulent.

Etiology.—(1) A perforating wound of the chest, as by a spear or sword thrust, or a bullet wound, may permit air to enter the pleural sac, and we may include in the same group cases in which, from cancerous or other ulceration, the œsophagus or stomach becomes connected with the pleural sac. (2) In many traumatic cases the visceral pleura is torn by a fractured rib, and so permits of the escape of air. Cavities of tubercular origin, if situated near the pleural sac, not infrequently ulcerate into it, forming a common non-traumatic cause of pneumothorax. In cases where bullæ or blebs occur in emphysema, one or more of these may give way and cause a pneumothorax, which, unless associated with pleuritic effusions, may become generalised. (3) In a small proportion of cases gas forms in the pleural sac, and generally in connection with exudations previously present. The organism usually responsible is the *Bacillus aerogenes capsulatus*.

Pathological Anatomy.—A pneumothorax may be limited by adhesions, and this is frequently the case where tubercular cavities ulcerate into the pleural sac. The opening into the pleural sac, if made by a fractured rib, or by a dagger or sword thrust, may cause a valvular opening, so that the air can freely enter the pleural sac from the lung, but cannot escape during expiration, with the result that the pressure in the sac may rise far above the pressure of the external air, and cause alarming dyspnoea and great displacement of the mediastinum and heart, and also of the diaphragm. In many cases in which fluid and air are present together, the fluid is purulent, and the condition is termed *pyopneumothorax*.

This is a condition of the pleura similar to what obtains in empyema, with, in addition, an opening either internally into the lung, or externally, by which opening the air has entered.

The lung collapses, the extent depending on the effusion, and in cases of pyopneumothorax the affected lung may not merely be collapsed, but is often bound down by firm pleuritic adhesions.

Clinical Features.—Sudden severe pain, with increasing dyspnoea, marks the development of a pneumothorax, and even where it is limited in extent, as it may be in cases due to a tubercular cavity, the dyspnoea present is suddenly increased, cyanosis becomes marked, and the pulse feeble and rapid.

The *Physical Signs* of a case of pneumothorax involving the whole or a great part of one side of the chest are very typical. On *Inspection* there is great distension, with immobility of the affected side, the lower interspaces being widely dilated, and the diaphragm depressed. On *Palpation* the vocal fremitus is diminished or lost; on *Percussion* the note is tympanic, and where there is an opening into the lung or externally through the chest wall, a very forcible stroke may elicit a somewhat cracked-pot resonance. Where the pressure rises to a very great extent in the cavity, the note got on percussion may become almost dull. *Auscultation* reveals loss of breath murmurs, and sometimes of vocal resonance, while consonating râles are only heard where moist râles are present in the lung, and fairly near the ear.

There are three phenomena which are typical of pyopneumothorax.

(1) *The coin test*, or *bruit d'airain* described by Trousseau, is got by tapping one coin used as a hammer on a second coin placed on the chest over the cavity, and at the same time auscultating over the cavity, the ringing sound is got where the coin-tapping and the stethoscope are *both* over the air-containing space. By this means the cavity may be sharply mapped out, and more accurately than by percussion alone. The coin test is also applicable to large cavities in the lung, which are near the chest wall and of sufficient size. For the sake of practice the student is advised to carry out this mode of examination on the normal stomach.

(2) *The metallic tinkling of Laennec* is typical of a cavity in the lung, as well as of a mixture of pus and air in the pleural

sac. It is believed to be due to the bursting of small bubbles of air in the cistern of fluid: it is sometimes got by shaking the patient, and it has been suggested that after the patient has been shaken, drops of pus may fall from the roof of the cistern into the fluid below, and thus give rise to the tinkling sound described by Laennec. The characteristic tinkling depends on the production of over-tones in the cavity.

(3) *Hippocratic succussion* is got by violently shaking the patient, and at the same time applying the ear or stethoscope to the affected side, when splashing of the fluid is audible. This phenomenon cannot be got in an ordinary pulmonary cavity.

In a general pneumothorax the enormous displacement of the heart and the diaphragm must not be forgotten; this displacement is not so great where there is a free communication between the outer air and the pleural sac.

The **Diagnosis** is easy unless the pneumothorax be very limited, when it may be impossible to discriminate between a large superficial lung *cavity* and a limited pneumothorax.

The **Prognosis** depends entirely on the cause of the condition. In the case of a fractured rib or an incised wound, or the rupture of an emphysematous bulla, recovery is by no means improbable; the air acts as a splint, and may cause no trouble unless it contains septic organisms. In many tubercular cases a pneumothorax hastens the death of the patient, and cannot be looked upon as other than a very grave complication, death sometimes supervening in a few days or even a few hours after its occurrence.

Treatment.—Where fluid and air are present, resection of the ribs should be carried out, the surgical treatment for empyema being resorted to. In the case of a broken rib or an emphysematous bulla it is probably wise to leave the air alone unless the intrathoracic pressure rises too high, when tapping with a fine needle may have to be done to draw off some of the air. Keep the patient at perfect rest, and enjoin shallow breathing, in the hope that the air acting as a splint may hasten the healing of the damaged pleura, after which the air will rapidly become absorbed. Some authorities have suggested that either sterilised air or nitrogen may be used to replace the air already in the pleural sac, but as any harm will have already been done, it seems to be a totally unnecessary inter-

ference with the patient. A carefully applied bandage often greatly adds to the comfort of the sufferer, and a scrupulous watch should be kept for the first appearance of subcutaneous emphysema, which, however, may not require any special local interference.

Pain may call for the use of morphia; otherwise careful dieting is alone sufficient in most cases.

(7) TUBERCULAR INVOLVEMENT OF THE PLEURA

Wherever there is extensive tubercular disease of the lung, there is always a tendency for the appearance of tubercles on the pleura, and in cases of miliary tuberculosis it is common to find the visceral pleura almost as markedly affected as the lung itself. In some cases of miliary tuberculosis involving the pleura a hæmorrhagic effusion is present.

It may be stated again that in many cases pleurisy with effusion, whether sero-fibrinous or purulent, is associated with tubercle.

(8) TUMOURS OF THE PLEURA

In many cases of tumours involving the lungs, the pleura suffers to a greater or less extent, and not infrequently the effusions, sometimes hæmorrhagic, are associated with such pleural involvement. It is not common to find the pleura secondarily invaded before the lung. Occasionally a pleural endothelioma is met with.

XXI. AFFECTIONS OF THE MEDIASTINUM

TUMOURS

HARE's table of 520 cases of mediastinal disease is most instructive, and a large proportion are tumours. Of these, 134 were cancer, 98 were sarcoma, mostly primary, 21 were lymphoma, 7 fibroma, 11 dermoid cysts, and 8 were hydatids.

The **Clinical Features** of an intrathoracic tumour are sometimes very distinctive, but in other cases there may be absolutely nothing of diagnostic value. Dyspnœa, cough, and difficulty in swallowing are common symptoms.

Physical Signs.—On *Inspection* and *Palpation* dilatation of veins may be noted, and especially if either the superior vena cava or the left innominate vein is compressed. In the latter case the left side of the neck and left arm are œdematous. There is sometimes local bulging and possibly erosion of ribs or sternum. The tumour may pulsate, but is not expansile. It may displace the heart, and very generally interferes with the lungs by compressing one or other bronchus. *Percussion* often yields a definite localised area of dulness, and *Auscultation* may be negative, or the heart sounds may be well conducted, or the stridorous breathing with evidence of a septic pneumonia may point to compression of a bronchus. A pleural effusion is not uncommonly associated, and there may be symptoms of pressure on the œsophagus, or on some of the nerves.

Special care should be taken to palpate each rib separately, and to examine the sternum, as small secondary growths may sometimes be recognised.

The thymus gland, when involved, may form a tumour palpable in the episternal notch, or may erode the manubrium sterni.

The **Diagnosis** is obscure in the absence of definite symptoms and physical signs, but it is as a rule easy to differentiate between an *aneurism* and a solid tumour.

The following points are helpful :—The age, usually 35-45 in aneurism, at any time of life in tumour; the sex, usually male in aneurism, and either sex in tumour. Hard work suggests aneurism. There is no tracheal tugging in tumour, and no expansile pulsation. The pressure of an aneurism is a “living” pressure, or one that is ever varying; the pressure of a tumour is constant. Potassium iodide affords much relief in aneurism, but usually no relief at all in tumour.

The **Treatment** must be purely palliative and on general principles.

Enlargement of mediastinal glands is often of tubercular nature, sometimes abscess formation occurs from tubercular or other causes, and very frequently along with adherent pericardium there is a mediastinitis associated.

DISEASES OF THE URINARY SYSTEM

I. ABNORMALITIES OF THE KIDNEY

(1) MOVABLE KIDNEY ; FLOATING KIDNEY ; NEPHROPTOSIS

NORMALLY, both kidneys are kept firmly applied to the posterior abdominal wall by the peritoneum, by their blood-vessels, and by a considerable amount of fat, which generally surrounds each of these organs. Should the kidney have a definite mesentery (mesonephron), there will be an amount of movement proportionate to the length of that mesentery. It also stands to reason that where a considerable amount of fat has surrounded the kidney, emaciation may lead to corresponding mobility ; it is probable that the lifting of heavy weights, the enlargement of the kidney sometimes due to a tumour, and loss of the support of the abdominal wall, such as occurs after pregnancies, peritoneal effusions, and abdominal tumours, may lead to a certain amount of mobility of the kidney.

It is well to retain the term "*floating kidney*" for those cases in which there is a well-marked mesonephron, and to describe as "*movable kidney*" cases in which the kidney is palpable, but with no wide range of movement.

Etiology.—Many of the etiological factors have already been suggested. Women suffer more commonly than men, 80 to 90 per cent of all cases occurring in females. The age is generally from 25 to 40 years, and statistics appear to show that the right kidney is more frequently affected than the left, while in about one-sixth of the cases both kidneys suffer equally. Many authorities consider that tight-lacing has much to do with the development of floating kidney in women, and this may explain also its greater frequency on the right

side than on the left, from the relationship of the right kidney to the liver.

Clinical Features.—In *movable* kidney, there may be no symptoms, and the condition may not be discovered until the abdomen is accidentally palpated. In other cases, there may be dragging pain in the lumbar region, sometimes described as neuralgic, but the chief symptoms are referable to dyspepsia occurring in a neurasthenic patient. Constipation is frequently associated with the condition. Such movable kidneys are palpable, sometimes with ease, the bi-manual method greatly aiding in the recognition of the lower border of the kidney, and forcible pressure over the organ causes a peculiar sickening kind of pain which is very typical.

In cases of *floating* kidney, while there may be no symptoms, we not infrequently meet with severe attacks of renal pain due probably to twisting of the mesonephron; they may be associated with nausea, vomiting, and even collapse. These attacks have been termed Dietl's crises, after the writer by whom they were originally described. In these paroxysms the affected kidney is specially tender.

In floating kidney, one readily recognises on palpation the kidney-shaped tumour which passes freely downwards below the hand, when the patient takes a deep breath, and the condition is generally easily recognised.

Dilatation of the stomach has been described as being present in a percentage of cases of movable and floating kidney.

Diagnosis.—The shape of the kidney, where it is definitely floating, prevents any possible error. The severe attacks of pain described by Dietl and associated with floating kidney certainly resemble *renal colic*, *biliary colic*, and sometimes the pain met with in *cancer*, but the history of the case, and the absence of the typical clinical features due to biliary and renal colic, generally prevent any mistake, and once the pain has subsided the kidney is easily palpated.

The **Prognosis** is favourable in most cases.

Treatment.—In many cases of *movable* kidney it is only necessary to advise the patient to wear a bandage, sometimes with a suitable pad, and to recommend the avoidance of severe physical strain, such as lifting heavy weights.

In *floating* kidney, while sometimes the bandage and

pad may be amply sufficient, operative procedure is often advisable. It is better not to excise the kidney, but nephroraphy, by which is meant the stitching of the kidney to its attachment posteriorly, has proved very successful. For the attacks of pain morphia, local fomentations, and confinement to bed may be necessary.

Not infrequently there is a marked neurasthenic element which demands treatment, not merely for what may be termed the constitutional element present, but also for the accompanying dyspepsia.

(2) MALFORMATIONS OF KIDNEY

Malformations are as a rule unimportant, unless when operative interference, likely to interfere with the functions of one kidney, is under consideration. The following variations in size, shape, number, and situation may be briefly mentioned.

(1) *Size*.—There may be one large kidney practically taking up the duties of both; the second kidney may be absent altogether or extremely small in size.

(2) *Shape*.—A horseshoe-shaped kidney is not an infrequent abnormality; in it both kidneys are fused into one, the junction between the two being generally below. Sometimes the pelvis of the kidney with the ureter is in an abnormal situation.

(3) *Number*.—As already indicated one kidney may alone be present, sometimes both kidneys are fused together, as in the horseshoe-shaped kidney. Absence of both kidneys is not compatible with life, and need not be considered here.

(4) *Situation*.—One or both kidneys may be displaced, sometimes even being found situated in the pelvis, but very often there is merely increased mobility of one or both organs, the condition described under movable or floating kidney.

Occasionally the ureters are blocked, and interference with a ureter may cause hydronephrosis. Sometimes there is great dilatation of one or both ureters, the result generally of some obstruction. Abnormal arrangement of the ureters, both as regards number and site of entrance into bladder, is rarely of much clinical significance.

II. CERTAIN ABNORMAL CONSTITUENTS OF THE URINE

(1) ALBUMINURIA ; ALBUMIN IN THE URINE

Type 1.—Functional Albuminuria.—This is apt to occur (1) after hard physical work, (2) as the result of fright, (3) following in some persons on cold bathing, and (4) as the result of certain kinds of dyspepsia. In all of these cases the amount of albumin is small, and in many of them it is only present during a short period of the day. There may be tube casts, and if they are found along with the constant presence of albumin, there is generally a pathological change in the kidney, either in the walls of the glomerular capillaries, or in the lining cells of the glomerular tuft.

Neurotic albuminuria, which might be included under this type, occurs after epileptic fits, in tetanus, frequently in exophthalmic goitre, and after blows on the head.

Type 2.—Febrile Albuminuria.—Albumin is frequently present in many of the continued fevers, and especially pneumonia, diphtheria, and typhoid, and it probably is due to marked cloudy swelling of the tubular epithelium.

Type 3.—Toxic Albuminuria and Albuminuria due to Changes in the Blood.—The following are examples of this type : syphilis, scurvy, purpura, lead and mercury poisoning, and the presence of bile pigment or sugar in the urine. Ether and chloroform, when administered as anæsthetics, sometimes cause a temporary albuminuria.

Type 4.—Organic Albuminuria.—By this is meant the presence of albumin in the urine as the result of Bright's disease, definite congestion of the kidney, and sometimes disease of the pelvis of the kidney, or of some other part of the urinary tract.

(a) In acute and passive congestion of the kidney albumin is present, the former condition being a stage of Bright's disease ; included in this group are all forms of acute and chronic Bright's disease.

(b) In fatty and waxy kidney albumin is present, and also in cases of suppurative nephritis and tumour of the

(c) Very small amounts of albumin are present in primary cirrhosis of the kidney.

(d) The urine contains albumin in disease of the pelvis of the kidney, of the ureter, and of the bladder, and in these cases there is almost always pus present.

(2) HÆMATURIA, OR BLOOD IN THE URINE

Etiology.—There is a wide range of diseases in which blood appears in the urine. It may be present in many of the malignant types of continued fevers, and sometimes in leucocythæmia. There are numerous *renal causes*, such as acute Bright's disease, acute congestion of the kidney resulting from toxic agents, and especially cantharides, turpentine, and carbolic acid, new growths and calculus, and especially new growths situated in the calyces of the kidney. Sometimes in primary tuberculosis of the kidney, and commonly in cases of renal infarction, hæmorrhage occurs; while in *Bilharzia hæmatobia* and the *Filaria sanguinis hominis*, hæmaturia is the rule, and these diseases constitute common tropical causes of the condition.

From the *urinary passages* blood may also enter the urine, and especially in cases of calculi, *Bilharzia*, and tumour, and reference must be made to villous growths in the bladder, which so frequently cause repeated and serious loss of blood. In not a few cases of severe abdominal injury the kidney, ureter, or even the bladder may be damaged, and as a result blood may appear in the urine.

Occasionally what has been termed renal epistaxis occurs; this is generally a vicarious form of menstruation.

Diagnosis.—Much may be learned as to the origin of the hæmorrhage by studying the urine and the time when the blood appears. From the *kidney* the blood is intimately mixed with the urine, which is of a smoky appearance. From the *pelvis of the kidney*, as for example where small tumours, frequently of malignant origin, bleed into the calyces or pelvis of the kidney, there is apt to be clotting of blood in the ureter, and consequent passage of small casts with the pains of renal colic. In hæmorrhage from the *bladder* the blood is passed with the terminal portion of the urine, and from the *prostate*, bleeding is apt to occur at the end of

micturition. From the *urethra*, blood comes at the commencement of the flow of urine.

Sometimes the examination of the cells present in the urine may yield important information, and especially if these cells should be of a malignant nature. Cystoscopy and the differential examination of the urine from the two kidneys often give important information as to the site of the hæmorrhage.

The **Prognosis** and **Treatment** naturally depend upon the causal conditions present, and will be referred to under their respective heads.

(3) HÆMOGLOBINURIA

The presence of blood pigment, hæmoglobin, or methæmoglobin in the urine, with very few blood corpuscles or with none at all.

Etiology. — (1) *Toxic Hæmoglobinuria*.—This is due to poisons which produce rapid destruction of the red blood corpuscles, and these include chlorate of potash, carbon monoxide, carbolic acid, strong mineral acids, such as hydrochloric acid and sulphuric acid, pyrogallie acid, naphthol, muscarine, and sometimes also the toxins of scarlet and other fevers. Exposure to excessive cold, great muscular exertion, a severe burn, and the administration of quinine to malarial patients, have all been occasionally followed by this condition. It is a well-known fact that the blood of one animal transfused into another may lead to hæmoglobinuria, and in the newly born there is an epidemic form associated with jaundice and certain nerve symptoms.

(2) *Paroxysmal Hæmoglobinuria*.—This is found especially in Raynaud's disease, and it is reputed to occur also, though rarely, in malaria and syphilis. The urine is reddish-brown or black, and has a thick reddish-brown deposit. There may be débris of red blood corpuscles, renal epithelium, and often a copious deposit of urates, while the urine always contains albumin.

Clinical Features.—The spectroscopic test for methæmoglobin is probably one of the most accurate. There are two absorption bands between D and E (Frauenhöfer's lines), and a deep, much more definite one between C and D.

In the *toxic* type of the disease the patient yawns, is excessively languid, frequently vomits, suffers from diarrhoea,

and may pass speedily into stupor and death. Occasionally definite rigors are present.

The *paroxysmal* type is not, as a rule, fatal. It begins much like the toxic type, and often with rigors. There is vomiting, diarrhœa, and pain in the lumbar region, while abdominal discomfort is extremely common. There may be several paroxysms in the same day, but as soon as the paroxysm has passed off, the urine becomes clear. During the attack there is often frequency of micturition, and jaundice is a usual sequel.

The **Prognosis** depends on the nature of the case, and, as already stated, the paroxysmal form is not dangerous, like the toxic type, unless nephritis is superadded; but it must not be forgotten that tubules and glomeruli may be blocked with blood pigment. Paroxysms may recur for years, and render the patient very anæmic.

Treatment.—Direct the patient to avoid any cause known to produce the paroxysmal attacks. Recommend warm clothing, no exposure to cold, and residence in a warm climate. During the attack give warm drinks and wrap up the patient warmly, while sedatives such as hyoscyamus, and astringents such as acetate of lead and gallic acid, may be tried with advantage. In certain cases adrenalin and similar hæmostatics have been administered, and cold may be applied, in place of heat, over the region of the kidneys. In syphilitic cases, give the usual antispecific treatment.

(4) PYURIA; PUS IN THE URINE

Where the pus is of urethral origin, it appears with the first portion of the urine. Where it is due to cystitis, it is apt to be passed in considerable quantity towards the end of micturition. In cases of cystitis, the urine is sometimes acid and sometimes alkaline; in men it is generally the latter, and contains a copious phosphatic deposit, while in pyelitis, the urine is more frequently acid in reaction. Pus in acid urine remains as a heavy deposit separate from the urine at the bottom of the glass, while pus in alkaline urine forms a ropy deposit similar to what obtains when an alkali is added to pus in a test-tube.

In diagnosis epithelial elements should be carefully looked

for, although tailed cells may come from other parts of the urinary tract than the pelvis of the kidney. The presence of great numbers of vaginal squamæ cannot fail to be recognised where the pus is of vaginal origin.

(5) CHYLURIA

Chyluria occurs in cases of filariasis, and chyle is also sometimes present in the urine in cases in which there is no satisfactory explanation for its occurrence. It is, in these instances, almost invariably mixed with blood.

(6) GLYCOSURIA

There is nearly always a trace of sugar in the urine in health, but it is with its presence in excess that we have to do here. It should be remembered that there are agents, other than glucose, which reduce copper, for a full account of which the reader should consult a text-book on clinical medicine. Glycosuria is not uncommon in stout persons after middle life, and perhaps especially where there is a gouty element in the history. A temporary glycosuria is not infrequent after the inhalation of chloroform or ether in certain subjects; it is sometimes present in epilepsy, and sometimes during pregnancy, and very commonly it follows severe injuries to the head. Glycosuria which can be removed by dietetic treatment should not be termed true diabetes mellitus, and care should be taken to obviate, by careful qualitative methods of examination, mistakes due to reducing agents in the urine, such as alcapton and glycuronic acid, both of which may be present in certain instances.

OTHER CONSTITUENTS

Acetone, diacetic acid, and β -oxybutyric acid are all referred to in connection with diabetic coma, and do not require further notice here.

In rare cases of diabetes mellitus, fat has been found present in the urine.

III. NORMAL CONSTITUENTS OF THE URINE, BUT IN EXCESS

(1) EXCESS OF URIC ACID, LITHURIA, LITHÆMIA

THE conditions under which uric acid is precipitated are mentioned in connection with uric acid calculi, but it should be remembered that hyperacidity, a low pigmentary index, and poverty in mineral salts may produce a precipitate of uric acid which does not depend on any excess of uric acid in the urine. The reader is advised in connection with this subject to refer to the description of gout and the formation of uric acid calculi in that disease. It is only necessary to add here that some error of oxidation probably accounts for cases in which there is a true excessive formation of uric acid, and that excess in eating and drinking with insufficient exercise goes a long way towards the explanation of lithæmia. Associated with it there is not infrequently primary cirrhosis of the kidney with an arterio-sclerosis generalised throughout the arterial system.

(2) OXALURIA

It is doubtful whether oxalates appear in the urine from other causes than the ingestion of oxalic acid with the food; but where there is an excess of oxalate of lime present in crystalline form in the urine, there is nearly always a marked degree of dyspepsia with profound depression of spirits, constituting the form of dyspepsia referred to under chronic gastric catarrh, page 217. It is common in gouty persons, and is often associated with a deposit of uric acid. Many subjects of oxaluria are hypochondriacal and neurasthenic.

(3) PHOSPHATURIA

By this is meant not the presence of the normal acid phosphate of soda, but the alkaline phosphates which render the urine alkaline, and which are often precipitated as earthy phosphates. When urine decomposes, the breaking up of urea renders the urine alkaline, but the alkalinity is volatile, because

it is due to ammoniacal decomposition. Where, however, the alkalinity is fixed, it is generally due to a true excess of phosphates. This is certainly not uncommon in protracted ill-health, and perhaps especially in diseases of the nervous system, although it may be present with any debilitating illness. Cases have been described of phosphatic diabetes in which there is a great increase in the excretion of phosphates, but this is rather an excess of acid phosphates than alkaline. It is a rare ailment, and its exact significance is not clear.

IV. ANURIA

A CONDITION occasionally met with in various morbid conditions, and consisting in arrest of urinary secretion.

(1) **The Non-obstructive Form**

Etiology.—Acute Bright's disease, especially scarlatinal nephritis and acute congestion of the kidney, whether associated with local pyogenic organisms or not, may cause suppression of urine. It may occur in the last stage of chronic Bright's disease, in the cold stage of cholera, after any severe shock, as from an internal injury, and also in collapse, while a less grave reflex form of anuria sometimes follows catheterisation.

Clinical Features.—The symptoms are serious from the first—vomiting, purging, convulsions, and frequent rigors being present, and death may occur in two to three days, with all the symptoms of toxic poisoning.

Diagnosis and Prognosis.—The passage of a catheter demonstrates the absence of urine in the bladder, while by careful abdominal examination hydronephrosis and similar conditions must be excluded. Any urine which is passed is small in amount, and loaded with albumin, and usually contains blood and many casts. The prognosis is extremely grave unless prompt relief can be obtained.

Treatment.—A hot bath should be administered at once, and hot poultices or fomentations applied over the region of the kidneys, while the skin may be stimulated to vigorous action by hot-air or radiant-heat baths. Sometimes cupping over the kidneys and hot rectal douches are of great value.

(2) The Obstructive Form

Etiology.—This is generally the result of the impaction of a calculus in the ureters, or pressure on them by some morbid growth. Congenital malformation of the ureters, slight at first, may gradually cause increasing obstruction.

Clinical Features.—During the first few days of the obstruction the patient suffers little inconvenience; there is usually a flow, from time to time, of a little pale urine of low specific gravity and containing little or no albumin. By the end of a week uræmic phenomena develop—sickness, gradual loss of muscular strength, and muscular twitchings, while increasing vomiting and diarrhœa indicate the onset of the toxic phenomena which terminate much like the more serious non-obstructive form, unless relief is obtained. There may be a combination of drowsiness with marked delirium, total inability to eat, and profound prostration, and the phenomena practically correspond with what we meet with in uræmia.

Diagnosis.—It is often possible in hydronephrosis to recognise the presence of a renal swelling indicating the marked distension of the pelvis of the kidney, while the passage of a clear limpid urine in diminishing amount, coexistent with the onset of uræmic phenomena, is very suggestive.

Prognosis.—Death occurs in 9 to 11 days, unless relief is afforded by surgical treatment.

Treatment.—In a case of calculus, an attempt may be made by vigorous abdominal massage (the patient being under an anæsthetic) to displace the stone, while the sedative effects of a hot bath and of tincture of hyoscyamus may permit of the relaxation of spasm, and so allow the calculus to proceed downwards. Where such treatment fails, an abdominal operation is indicated, as it offers the only chance, in a large majority of cases; but it may be found impossible to give the patient permanent relief in tumour cases.

V. URÆMIA

A TOXIC condition of the blood not infrequently found in chronic Bright's disease, and from which a considerable per-

centage of patients so affected eventually die. It is also the result of anuria. Unfortunately we cannot state, with certainty, what is the exact nature of the toxic substance or substances. The following theories have been put forward to explain the condition:—

1. *Toxic Agents*.—(a) Substances retained in the blood which ought to be excreted by the kidneys; (b) Abnormal decomposition in the blood, or possibly in the tissues, of such retained substances; (c) The formation of abnormal products of metabolism by the tissues.

The toxic agent does not seem to be urea, because urea can be administered experimentally without the production of uræmia. The blood may contain a considerable amount of nitrogenous extractives, principally urea, without the production of uræmia. Normally there is $\cdot 015$ per cent of urea in the blood, while in renal disease without uræmia there is $\cdot 15$ per cent of urea, in addition to a considerable amount being excreted by the kidneys. In acute uræmia the percentage rises to $\cdot 45$ or $\cdot 5$ per cent. While, therefore, the urea alone cannot be considered the sole cause of uræmia, the examination of the amount of urea excreted in the urine and the investigation of the amount of urea present in the blood yield definite information with regard to the probable onset of uræmic poisoning. A large amount of urea present in the blood indicates a distinct risk, and a marked diminution of the urea excreted by the kidneys is also associated with a tendency to uræmia. In making such calculations it must be borne in mind that the amount of urea and nitrogenous extractives excreted by the kidneys varies directly with the dietary, and allowance must be made for a further diminution in cases where the patient is confined to bed.

2. *Edema of the Brain*.—This is commonly present post mortem, but it is by no means associated with uræmia alone.

Etiology.—Uræmia may develop in chronic Bright's disease, less frequently in the acute forms, also in cirrhosis of the kidney, in active and passive congestion of the kidneys, in waxy degeneration, in tubercular disease, in calculus, in hydronephrosis, in cystic disease of the kidneys, and in association with suppression of urine where no kidney disease may be present at all. It should be stated that while the diseases

above mentioned predispose to uræmia, there is often, in addition, some exciting factor, such as exposure to cold, over-work, imprudence in diet, or, in short, anything which throws a special strain on the renal organs.

Clinical Features.—There are various types, each consisting of a group of symptoms which may be described separately, although a combination of these is not uncommon.

Type 1. Convulsions.—These come on suddenly, generally with headache and great restlessness. The convulsions resemble Jacksonian epilepsy, or sometimes an ordinary epileptic seizure, but in place of one, or perhaps two fits occurring, there is a tendency to the status epilepticus; in the intervals between the fits the patient is practically unconscious, or else in a few isolated cases he may be highly excited and almost maniacal. Associated with these convulsions are marked twitchings of muscles seen between the fits, and often indicating that a new seizure is about to occur. Temporary blindness and deafness are sometimes present with uræmic convulsions, while headache, sleeplessness, and alimentary disturbance may be noted in greater or less degree.

Type 2. The Maniacal Type.—In this form there is intense restlessness and mental excitement, with absolute sleeplessness and eventually wild delirium. The attack may develop suddenly, and is apt to be rapidly fatal.

Type 3. The Dyspnoeic Type.—This type resembles a form of asthma, the breathing, however, being of a peculiarly hissing character. There is marked orthopnoea, and in certain cases the breathing becomes Cheyne-Stokes in character. Sometimes the pupils contract and dilate rhythmically, and the pulse may alter in speed. Associated with this type there are often some of the other manifestations of uræmia.

Type 4. The Gastro-intestinal Type.—In this type there is great nausea and incessant vomiting, often associated with severe abdominal pain. Hiccough, of a most persistent nature, commonly occurs, and intractable diarrhoea may be present. In certain cases the diarrhoea occurs without any gastric disturbance, but this is rare.

Type 5. The Comatose Type.—With or without preceding delirium the patient becomes comatose. There may be initial cramps or twitchings of muscles or gastro-intestinal phenomena, but with the advent of coma the patient soon dies. Probably

this type is one of the commonest, and often constitutes the final stage of an acute uræmia.

Type 6. The Paralytic Type.—In certain cases of uræmia, perhaps more frequently in the chronic than in the acute form, a monoplegia or a hemiplegia develops, while there may be no hæmorrhage or other gross lesion in the brain to account for its presence. Such cases are by no means uncommon, and their explanation is as yet unknown.

While uræmia has been described under these different types, many cases show phenomena referable to several or possibly all of them, and it is well to remember that the most common features of any uræmic attack are the following—gastro-intestinal irritation, headache, uræmic dyspnœa, twitchings of muscles, and a distinct tendency to convulsive seizures, mental excitement, and coma. No reference has yet been made to pyrexia; sometimes there is a marked degree of temperature, although it is possible that it may be due, in not a few cases, to a pleurisy, pericarditis, meningitis, or other complication, rather than to the toxæmia.

Not infrequently a form of uræmia which might be designated *chronic* occurs, and although the symptoms are similar to those just mentioned they are less acute. They consist in headache, twitchings, attacks of dyspnœa, often rather suggestive of asthma and nocturnal in occurrence, restlessness and sleeplessness, sometimes somnolence and stupor, itching of the skin, and a varying amount of nausea, vomiting, and diarrhœa.

Diagnosis.—The quantity of urea in the blood is probably one of the safest guides to the diagnosis of uræmia. The following conditions are liable to be confused with uræmia:—

(1) Occasionally a *continued fever* may be mistaken for uræmia, and perhaps especially *typhus* and *typhoid*, and it may be extremely difficult, unless after a careful consideration of the symptoms and signs, to offer an absolutely certain diagnosis. Unquestionably the presence of an excess of urea in the blood would be an important point.

(2) *Opium poisoning* and *alcoholic poisoning* may be mistaken for the coma met with in uræmia. Generally the presence of albumin in the urine is of the greatest importance, while the odour of the patient's breath may, on the other hand, suggest opium poisoning or alcohol. The pin-point

pupils in opium poisoning and the dilated pupils in alcoholic stupor may both be simulated in uræmic coma.

(3) Uræmic paralysis is sometimes extremely difficult to diagnose from a *monoplegia* or *hemiplegia* the result of an organic brain lesion. The examination of the blood and the urine may help in obscure cases.

If albuminuric retinitis is present, there is positive evidence of kidney disease, and in all doubtful cases an examination should be made of the condition of the fundus.

Prognosis.—Acute uræmia of any type is apt to be fatal, whereas the more chronic forms, although they may eventually lead up to a fatal result, rarely directly cause it.

The duration of life in a patient who has once had uræmia will depend largely on the state of the kidneys, and on the possibility of careful treatment, medicinal and dietetic, but so many cases terminate fatally during the first attack that the prognosis must always be grave.

The **Treatment** is practically that described in Bright's disease in its different forms, especially the use of purgatives and diaphoretics.

For the convulsions chloroform anæsthesia is often advantageous, and, where the vascular tension is high, an attempt may be made to reduce it by means of nitroglycerine (gr. $\frac{1}{100}$ – $\frac{1}{50}$), by careful attention to the free action of bowels and skin, and perhaps above all by a well-regulated and suitable non-nitrogenous dietary. Diaphoresis may be secured by the hot-air bath and pilocarpine administered in $\frac{1}{2}$ th to $\frac{1}{6}$ th grain dose hypodermically.

VI. ACUTE CONGESTION OF THE KIDNEY

THIS is an early stage of acute Bright's disease, but it may also be the result of the following conditions:—

1. The action of irritant substances on the kidney, such as excessive amounts of cantharides, carbolic acid, turpentine and copaiba, quinine and mineral acids.

2. Toxins produced by organisms and excreted by the kidneys, and also the presence of certain organisms themselves. Probably the first stage of Bright's disease is often due to the presence of toxins.

3. Active congestion from dilatation of vessels. This is

a doubtful cause, but it may possibly occur through the agency of the nervous system as a reflex nerve action, and it has been thought that by this means the renal congestion following catheterisation, and perhaps that following exposure to excessive cold, may be brought about. Experimentally it has been found that stimulating the central ends of the lower dorsal roots produces acute congestion of the kidneys.

Pathological Anatomy.—The kidney is swollen and engorged with blood, the glomeruli are bright red in colour, and there are frequently hæmorrhages into them or into the tubules, while the tubular epithelium is swollen, showing cloudy swelling, and may even have reached the further stage of desquamation.

The **Clinical Features** resemble the early stages of acute Bright's disease with the presence of albumin, frequently blood, and generally epithelial and hyaline casts in the urine.

The **Treatment** is directed to the removal of the cause of the condition, and will be referred to further under acute Bright's disease.

VII. PASSIVE CONGESTION OF THE KIDNEY

Etiology.—It may be the result of heart disease, and of adherent pericardium, and it commonly follows in the train of such pulmonary conditions as bronchitis, emphysema, and phthisis. It may also be due to obstruction of the inferior vena cava, and may occur in cases of ascites, especially where, as the result of cirrhosis of the liver, there is interference with the venous circulation. Malignant growths sometimes infiltrate the renal veins, and thrombosis of these veins may occur in debility.

Pathological Anatomy.—The kidneys are large in size and dark red in colour. They are hard and firm, and engorged with blood, and the stellate veins are well marked. Interstitial changes are common in long-standing cases, and may be associated with thickening of vessels and of Bowman's capsules.

Clinical Features.—There is often a reduced quantity of urine (10-20 oz.), and it is generally of high specific gravity, of a dark colour, and throws down a uratic deposit on cooling. The albumin varies in quantity, being rarely large unless inflammatory changes are present in addition. There are hyaline and sometimes granular casts.

Diagnosis.—Is the albumin simply the result of passive congestion, or is there an inflammatory process present in addition? The albumin is invariably limited in passive congestion, and a large amount of albumin together with epithelial casts would suggest nephritis. An attempt should be made to discover the cause of the passive congestion.

The **Prognosis** depends on the possibility of relieving the backward pressure, and is distinctly more favourable if there is no inflammatory condition present.

Treatment.—Treat the heart, lungs, or other causal factor, and administer hydragogue purgatives and diuretics in almost every case. Much may also be done to afford relief by poulticing or dry-cupping the loins.

VIII. ACUTE NEPHRITIS

(ACUTE BRIGHT'S DISEASE)

AN acute inflammation of the kidney, which may involve especially (1) the glomeruli, (2) the convoluted tubules, or (3) the interstitial tissue; in every case, although one or other of these may be chiefly affected, all suffer.

Etiology.—There are probably predisposing as well as exciting causes in most instances, and it is difficult to assign to each the due amount of blame.

1. Cold and wet are extremely common causes, and especially where perspiration is suddenly checked, or where alcoholic subjects or debilitated persons are exposed to sudden and great changes of temperature.

2. In some of the specific fevers, and especially in scarlet fever, small-pox, and yellow fever, and less frequently in typhoid, cholera, and malaria, acute nephritis may supervene as a complication. It may be due to the toxin, and perhaps, in certain cases, to the presence of the organisms themselves where they are excreted by the kidney. Associated with this group is the incidence of acute Bright's disease in cases of septicæmia, pyæmia, erysipelas, and acute tuberculosis.

3. Certain poisons, such as cantharides, turpentine, carbolic acid, and large doses of strong mineral acids, may produce acute nephritis; while lead and probably long-continued and oft-

repeated smaller doses of the drugs just mentioned are capable of causing a more chronic form of the disease.

4. Certain substances excreted by the kidney may act as toxic agents; for example, sugar in diabetes mellitus and bile in permanent jaundice.

5. In pregnancy, acute Bright's disease may supervene, perhaps as the result of some unknown toxin or from special strain being thrown upon the kidneys.

6. Acute Bright's disease may also follow extensive burns, especially of the trunk, persistent eczema and erythema involving large areas of the skin. Possibly this group depends on absorption of some toxin or toxins, but it may also be due to reflex nerve stimulation.

7. Acute Bright's disease may result from a renal operation, or injuries affecting the region of the kidney, and may be due to the introduction of some organism, or in certain cases to congestion from reflex irritation.

Acute Bright's disease is common in childhood and up to the age of 40, but is rare after middle life. It is more common in men than in women, the proportion being as 3 to 2.

It is doubtful whether heredity has much to do with a predisposition to Bright's disease, but certain families in successive generations have supplied an unusual percentage of cases. This probably depends rather on inherited weakness of constitution than anything else. Undoubtedly debility both of mind and body and intemperance predispose to acute Bright's disease.

Bright's disease is more common in temperate climates.

Pathological Anatomy.—The appearance of the kidneys varies much according to whether the glomeruli, the tubules, or the interstitial tissue have specially suffered, and the congestion and inflammatory infiltration may be more or less localised to one or other of these structures, although it is often generalised. Where there is much interstitial exudation, there is considerable enlargement of the organ. The kidneys may be congested and engorged with blood, or the cortex may be pale and swollen, while the medulla is red and hyperæmic. The capsule strips freely.

(1) The glomeruli, when they are specially affected, may be seen with the naked eye as bright red points in the cortex. On microscopic examination hæmorrhages are frequently seen

within Bowman's capsules, and the epithelial cells covering the tuft proliferate and begin to desquamate, while leucocytes may also be observed in considerable numbers. Sometimes the capsules and the capillaries show hyaline changes in their walls.

(2) The convoluted tubules, where they are chiefly affected, show marked cloudy swelling with commencing desquamation of the renal epithelial cells. The desquamated cells are seen to contain droplets of fat when examined with the microscope, while the nuclei lose their distinctness, and red blood corpuscles and leucocytes may be found inside the tubules.

(3) The interstitial changes consist of an exudation of inflammatory lymph with red blood corpuscles and leucocytes, specially between the convoluted tubules. These changes are frequently more or less localised to the neighbourhood of the larger vessels supplying the cortex, and may vary considerably in amount in different parts of the same kidney.

All toxic cases tend to show marked *glomerular* inflammation. Acute nephritis, the result of scarlet fever, of cantharides, or of carbolic acid poisoning, is a good example of this variety, while exposure to cold and wet produces chiefly *tubular* changes associated with a considerable amount of *interstitial* exudation. It must be remembered that these statements are by no means always correct, and that following on glomerular changes the tubules may be secondarily involved, and interstitial exudations may also occur. Swelling of the cortex depends largely on the involvement of tubular epithelium and on interstitial exudations, and in these cases the cortex of the kidneys is frequently swollen but pale. When interstitial exudation has been replaced by the formation of connective tissue, the convoluted tubules are liable to be compressed and local dilatations occur.

In the medulla of the kidneys in acute Bright's disease there is also congestion, frequently hæmorrhages, and more or less small-cell infiltration, although it does not suffer to the same extent as the cortex.

Clinical Features.—*Type 1. Acute Nephritis, the Result of Exposure to Cold or Wet.*—The onset is generally sudden, œdema or puffiness of the face, and especially of the eyelids, develops within a few hours, and on examining the eyes

œdematous fluid may be seen under the ocular conjunctiva, which has been aptly termed the "tear that never falls," and which gives to the eye a peculiar glistening expression. The ankles may also show dropsical effusion. There is usually pyrexia, and possibly rigors, the temperature reaching 101° or 102° F., while the patient suffers from considerable malaise and general discomfort.

Urinary System.—The urine at first is scanty, although micturition is frequent, and it contains albumin, blood, and hyaline, epithelial, and blood casts; later the urine becomes less "smoky," and contains a varying amount of albumin, often large, with much renal epithelium, and hyaline, epithelial, and granular casts. The urea diminishes to about half the usual quantity. On chemical examination both serum albumin and serum globulin are found to be present, and sometimes albuminoses.

Alimentary System.—There is often acute dyspepsia with nausea and vomiting, the appetite is poor, and in certain cases persistent diarrhœa supervenes. One of the serious complications of acute Bright's disease is peritonitis.

Hæmopoietic System.—Anæmia develops at an early stage owing to the waste of albumin, and the pale, pasty face of a patient suffering from acute Bright's disease is most characteristic. Sometimes epistaxis occurs.

Circulatory System.—In 3 to 6 weeks the cardio-vascular phenomena of Bright's disease may develop. There is often, to begin with, increased tension of the pulse owing to vasomotor spasm, the result of toxins circulating in the blood. At a later stage there is definite arterio-sclerosis with hypertrophy of the left ventricle. The complications in connection with this system include pericarditis and sometimes acute dilatation of the heart.

Respiratory System.—Edema of the lungs is common in Bright's disease, and may occur to a serious extent in acute nephritis, while bronchitis sometimes develops. The complications are pleurisy, not infrequently with marked effusion, and sometimes pneumonia.

Integumentary System.—The skin is dry and perspiration scanty. Sometimes eruptions appear, but they are not typical.

Nervous System.—Uræmic phenomena may develop; certain of these belong to other systems, and have already been men-

tioned. They are nausea and vomiting, twitching of muscles, severe headache, epileptiform convulsions, amaurosis, and sometimes a very marked dyspnoea not entirely explained by the amount of œdema of the lungs. Retinitis and papillitis are more commonly found in chronic than in the acute forms of Bright's disease.

Type 2. Post-Scarlatinal Nephritis.—While many of the symptoms are the same, the following points are worthy of special note:—The onset is more gradual, and convulsions are not infrequently present in children, while the temperature, which had probably fallen to normal, once more reaches 102° , or possibly 103° F. The degree of dropsy varies considerably, and so does the amount of blood in the urine. In other respects the description already given under type 1 applies also to the post-scarlatinal form of Bright's disease.

Diagnosis.—The dropsy, when present, and especially involving the face, is very characteristic, and if it is associated with a diminished quantity of urine and urea, a large amount of albumin, the presence of blood and of epithelial and other casts, the diagnosis becomes simple. In *passive congestion of the kidney* there is diminished urine, but the albumin is small in amount. It must, however, be stated that there are insidious cases of the disease in which uræmic phenomena may first suggest the presence of serious nephritis. Cases of acute Bright's disease without the presence of albumin in the urine have been described, but must be exceptional. The nature of the tube-casts offers one of the best methods of diagnosing the variety of Bright's disease from which the patient is suffering.

In pregnancy it is wise towards the later months to examine the urine on several occasions.

The **Prognosis** is often favourable, although the condition may tend to become chronic. An acute nephritis after exposure to cold or wet is more likely to terminate favourably than a post-scarlatinal case. The mortality in young children is high, and $\frac{1}{3}$ rd of all fatal cases occur in them.

The duration of a case of acute nephritis varies considerably, but from 1 to 3 weeks generally elapse before marked improvement occurs, while in other cases it may be 6 or 8 weeks before the urine becomes free from albumin. Generally the dropsy disappears first and the albumin later. In not a

in these cases, unfortunately, the persistence of the albumin indicates a permanent change in the kidneys.

Treatment.—The patient should be placed in bed between blankets, and a flannel night-dress should be worn. This is desirable, because free diaphoresis forms perhaps the most important method of treatment, and chills must be strictly avoided.

The *diet* must be limited to milk, and milk alone, and some authorities prefer buttermilk. Many patients require the addition of starchy foods such as arrowroot, sago, etc., but so far as possible meat and fish, even in the form of beef-tea and soups, should not be given. There are certainly a few cases in which a rigid milk or carbohydrate diet proves unsuitable, and to them a small quantity of beef-tea may have to be allowed. Oranges and lemons can be permitted, and saline mineral waters are of great value. As recovery occurs, spinach, cauliflower, and similar vegetables may be added to the dietary, and bread can be freely eaten, but it is prudent to avoid nitrogenous articles of food until the albumin has disappeared.

The following medicinal measures are useful:—

1. *Purgation.*—Saline purgatives are very suitable, such as magnesium sulphate (5 1-3), Carlsbad salts, Hunyadi Janos, and many purgative mineral waters. Amongst other hydragogue cathartics are compound jalap powder (gr. 20-30), and the pulvis elaterini compositus (gr. 1-4). Do not purge too freely unless uremia is threatening, because a severe form of anasarca is not uncommon in Bright's disease.

2. *Diaphoresis.*—Hot baths, hot-air baths in which the temperature may be raised from 120° at first to 170° or 180° F., and wet packs (hot water), are admirable methods of treatment. Jaborandi (tincture 5 ½-1) or pilocarpine hypodermically (gr. 1 ½-6) are the best drugs to use for stimulating diaphoresis, but it is desirable to investigate the amount of anasarca or edema present before using pilocarpine, as it may increase edema in a sudden and dangerous manner.

3. *Diuresis.*—It is certainly bad policy to stimulate an already overworked organ, and one object of eliminating waste matter by the lungs and skin is to save the kidneys as far as possible, but where uremia is threatening, or where dropsy is very extensive, it may be necessary to have recourse to diuretics. In

ordinary cases plain water or alkaline mineral waters form the best and simplest method of stimulating renal secretion, but the spirit of nitrous ether in 1-2 drachm doses may be administered occasionally, and often in association with the hot bath or hot-air bath.

The local treatment is very important, and especially if there is much lumbar pain and great hæmaturia. It consists in poulticing the loins and in very severe cases wet-cupping. A hot bath is an excellent method of treatment in children during the earlier stages of the disease as a means of relieving the congested kidneys.

There are certain conditions which demand special treatment; sometimes *dropsical effusions* require to be tapped, and Southey's tubes or Potain's aspirator may have to be used, the former for ascitic effusions and great œdema of the legs, the latter for hydrothorax. Care must be taken to prevent irritation of the skin, in view of the peculiar tendency of patients suffering from Bright's disease to succumb to the attack of the specific organism or organisms of erysipelas. *Vomiting* should be treated with dilute hydrocyanic acid and bismuth, and sometimes with minute doses of carbolic acid or iodine, while the *dyspepsia* so commonly associated with the disease may require the use of pepsin and hydrochloric acid. The *anæmia*, from which all Bright's disease patients suffer to a greater or less extent, should be treated with iron and sometimes with arsenic. Where albumin persists in the urine, various drugs have been recommended, although none of them appears to be of special efficacy. These are nitroglycerine in $\frac{1}{100}$ th of a grain doses, tincture of the perchloride of iron, which is supposed to act as a powerful astringent, and rosaniline, which has the effect of colouring the urine in a marvellous fashion, although it does not appear to produce any phenomenal benefit. A warm, dry climate is of great advantage in the after-treatment of acute Bright's disease, while special care should be taken of the patient, to guard against chills or against any strain being put on the kidneys by imprudent diet. Should dilatation of the heart occur, strychnine and arsenic must be carefully administered with prolonged rest in bed. The cardio-vascular changes associated with Bright's disease are referred to under cirrhosis of the kidney (p. 515).

IX. SUBACUTE PARENCHYMATOUS NEPHRITIS

Two varieties of Bright's disease may be included under this head: (1) the form which is the so-called "large white kidney," or more correctly the large type of subacute parenchymatous nephritis; and (2) a subacute variety of Bright's disease, in which the kidney is smaller in size, and which is either secondary to acute Bright's or commences as a less acute variety.

Type I.

Etiology.—It is generally of insidious development, and is sometimes due to excessive alcoholism, and sometimes to the three great causes of waxy degeneration, *i.e.* syphilis, chronic suppuration, and suppuration of bone. This form of Bright's disease may also be present in cases of tuberculosis, and possibly it may develop, in place of acute nephritis, after exposure to cold or after scarlet fever.

Pathological Anatomy.—The kidney is large, the capsule is not markedly adherent, probably in most cases stripping freely, the cortex is pale and swollen, and the pyramids may be deeply congested. Microscopically the tubules are distended, the epithelium is very fatty, the glomeruli are enlarged and their capsules thickened. The walls of the capillaries show evidence of hyaline degeneration, and the interstitial tissue is everywhere increased in amount. Where it is associated with amyloid degeneration, there are in addition marked waxy changes.

Clinical Features.—The urine is at first small in amount and very highly albuminous, although later polyuria may develop, especially in waxy cases; the urea is diminished, there is much degenerated renal epithelium, and many leucocytes, and hyaline, granular, epithelial, and fatty casts are present in the deposit. Dropsy of the face and body generally is typical, and the anæmia is profound, the face in particular being pale and pasty-looking. Gastro-intestinal symptoms are common, especially vomiting and diarrhœa, while ulceration of the colon may hasten a fatal result.

The cardio-vascular changes, while they may be present, do not develop so definitely as in chronic Bright's disease. Uræmia is of frequent occurrence and may cause death.

The **Diagnosis** between this form of Bright's disease and the second variety, to be mentioned immediately, is by no means easy. The insidious commencement, the frequent association with waxy disease, and the large number of fatty casts, suggest the probability of the large type of subacute kidney being present.

Prognosis.—Whether it is associated with waxy degeneration or not, the prognosis is always grave. Edema is common. Inflammation of serous membranes frequently occurs, and uræmia is a constant danger. The coexistence of waxy degeneration adds much to the gravity of the case.

The **Treatment** must be on general lines, and little can be done in the way of attempting a cure in these cases.

Type II.

In this variety of Bright's disease, which is much more common than Type I., the kidney is of smaller size, the cortex shows marked fatty changes, and, as a rule, fatty casts in the urine are numerous. The capsule is thickened and somewhat adherent. The amount of albumin is invariably greater than would be expected in a case of more chronic Bright's disease, but it is so closely associated with chronic Bright's disease, of which it is really a more acute variety, that its description will be given under that heading. Where a patient, who has suffered from acute Bright's disease, does not recover, there are apt to be frequent acute or subacute exacerbations, and after death marked fatty changes in the kidney are usually seen.

X. CHRONIC BRIGHT'S DISEASE

(CHRONIC INFLAMMATORY NEPHRITIS; CHRONIC INTERSTITIAL NEPHRITIS)

THIS form of Bright's disease may result from an acute case, or it may be chronic from the outset, although it is extremely unlikely that the type referred to as the large type of subacute parenchymatous kidney can ever become the "small white kidney" commonly met with in chronic interstitial nephritis.

Etiology.—The causes of acute Bright's disease may give rise to this variety, but probably toxic agents such as alcohol,

lead, and gout play a prominent part in the cases which are chronic from the outset.

Pathological Anatomy.—This variety of kidney disease is frequently termed the “small white kidney,” by which is meant that there are marked fatty changes in the cortex, and that the cortex is, as a rule, atrophied; but the degree varies with the development of interstitial tissue, and the kidney may therefore be, comparatively speaking, large, if it is the result of an acute or subacute affection, and may closely resemble the subacute parenchymatous kidney in appearance.¹ In many cases, however, the kidney is found to be small in size, closely resembling the small atrophied kidney of a primary cirrhosis, from which it may with difficulty be differentiated by the naked eye, although as the result of the marked cicatricial changes secondary to interstitial exudation the interlobular vessels become extremely tortuous, whereas in primary cirrhosis the vascular markings are usually straight. Further, in a post-inflammatory kidney, however advanced the cirrhotic changes may be, there is always greater evidence of fatty degeneration in the tubular epithelium.

The vessels are thickened, the thickening being greater in the more cirrhotic forms and the walls of the capillaries, and Bowman's capsules show marked hyaline degeneration. The tubules of the cortex are irregularly dilated. Much of the epithelium has been shed, and there is evidence of fatty degeneration, as already stated.

The capsule, in the cases where there is little interstitial change, strips more freely, although there are always some adhesions, but in the cirrhotic cases it is very adherent, and leaves a granular surface when it has been removed, which closely resembles what is seen in primary cirrhosis.

Clinical Features.—In cases previously acute, the history of the symptoms will correspond to what has already been stated under acute Bright's disease. In less acute cases, or in chronic cases, there may be an insidious development with gradual loss of strength, progressive emaciation, and profound anæmia. The degree of dropsy varies greatly; in certain cases it may be very marked, the face showing the typical puffy, pasty-looking appearance of an acute Bright's case, while in

¹ These cases are practically the same as, and certainly indistinguishable from, Type II. of subacute parenchymatous nephritis.

the more cirrhotic forms dropsy may be conspicuous by its absence. Similarly, in the urine, where the kidney is of larger size, the quantity of urine will probably be scanty and the amount of albumin large; on the other hand, in the "small white kidney" the urine tends to be excessive and the albumin less copious. The urea is generally diminished, and the casts vary both in character and number. There are always granular and hyaline casts, but fatty casts are specially frequent in the larger variety of kidney.

Alimentary System.—The gastro-intestinal phenomena are usually well defined, a degree of nausea and vomiting being almost constant, and the vomiting is generally of the type which occurs in the morning before breakfast. Hiccough is sometimes present and diarrhœa is not infrequent, and it should be remembered that enteritis sometimes ensues as a complication, and consequently diarrhœa adds much to the anxiety of those in charge of a case of chronic Bright's disease.

Hæmopoietic System.—Anæmia is the only prominent feature to be noted in this system.

Circulatory System.—Cardio-vascular changes are extremely marked, and especially in the small type of kidney. There is generally progressive, and often great, enlargement of the left ventricle, while the arterial walls are thickened and the pulse is of high tension. Partly as a result of these vascular changes, and partly on account of the presence of toxic substances in the blood, there is great tendency to hæmorrhage. Such hæmorrhages may occur from the gums, from the nose, from the kidney, and, not infrequently, as purpuric hæmorrhages, especially seen under the skin of the legs.

Respiratory System.—Dyspnœa is present if much pulmonary œdema has occurred, and asthma, in reality a uræmic manifestation, may also be noted.

Integumentary System.—The dropsy and purpuric hæmorrhages have already been referred to, and there is a peculiar liability to erysipelas where there is any irritation of the skin. Pigmentations are by no means uncommon, and eczema and sometimes erythema may be seen in certain cases.

Nervous System.—In referring to the nervous phenomena in acute Bright's disease we stated that many of them had already been mentioned under other systems, although they were in reality of uræmic nature, and the same statement

holds good here. The vomiting, nausea, and dyspnœa are all to a large extent of uræmic or, in other words, of cerebral origin. Headache is a prominent feature, and is dependent on the changes in the vessels, and perhaps partly on the high tension in the cerebral arteries, and it is noteworthy that after a severe attack of epistaxis or renal hæmorrhage, the headache is greatly relieved. Uræmic cramps and twitchings of muscles, Cheyne-Stokes breathing, and all the typical phenomena of uræmia referred to under that heading, are unfortunately only too common in chronic Bright's disease, and the slightest over-strain or over-exhaustion may precipitate a fatal attack; while the secondary inflammations of serous membranes which form such common complications in every variety of Bright's disease may readily induce uræmia. Albuminuric retinitis is specially apt to develop in all forms of chronic Bright's disease. It is seen as stellate patches of fatty degeneration round the macula lutea, and often there is marked papillitis with numerous flame-shaped hæmorrhages. As a result sight generally fails, and this may be the means of bringing the patient for the first time to seek medical advice. Cerebral hæmorrhage, while not so frequent as in primary cirrhosis of the kidney, is also apt to occur, and is often associated with the presence of small miliary aneurisms on the cerebral arteries.

Complications.—The only complications to which special reference is necessary are inflammations of serous membranes, and in particular of the peritoneum, pleura, and the pericardium, while mention may again be made of enteritis, a virulent type of which may terminate the patient's life.

Diagnosis.—There is little probability of a mistake where dropsy and a large amount of albumin are evident clinical features. Uræmic manifestations should not be forgotten, and a patient, supposed to be suffering from asthma, may be found to be really the subject of chronic Bright's disease. On examining the urine it is very unlikely that a *physiological albuminuria* could be mistaken for this disease, because the kind of casts usually found present and the amount of albumin are distinctive. In physiological albuminuria there is little albumin in most cases, and only a few granular or hyaline casts, and diet and exercise may be the factors which predispose to the presence of albumin at certain periodic times in the day. The greatest stress should be laid on the

careful examination of the heart and vessels, because cardiovascular changes inevitably follow in a well-defined case of chronic Bright's disease.

Prognosis.—The patient is invariably in a position of great danger, because the slightest overstrain may induce uræmia, and there is a considerable predisposition towards cerebral hæmorrhage. On the other hand, patients carefully treated and living under the best possible surroundings may enjoy long life. Dropsy is probably the feature which, above all others, with the exception of uræmia, is very grave.

Treatment.—Each case requires careful study. Find out the amount of the albumin, the number and variety of the casts, and the capacity of the kidney for carrying on the work of life. There are cases in which it is futile to attempt vigorous treatment with any hope of cure, and excessive bathing may only lead to serious exhaustion of strength.

A dietary as simple as possible should be rigidly adhered to, but it is unwise to limit the patient to milk alone. Specially forbid an excess of meat and all heavy and indigestible food, and particular stress should be laid upon the importance of abstinence from alcohol, or its use in very limited quantity. Anæmia should be treated by giving large doses of iron; plenty of fresh air, but without undue exercise or fatigue, is most important. Where dropsy is present, purgatives and diaphoretics are requisite, but, while care should be taken to see that the bowels act freely, it is unwise to over-stimulate the alimentary tract.

In cases of Bright's disease it is undesirable to use drugs which, owing to renal inadequacy, can accumulate in the system, and therefore opium should only be administered under exceptional circumstances. Mercurial salts should also be prescribed with caution, because in Bright's disease there is a special tendency to mercurialism.

Stripping off the capsule of the kidney has been recommended in cases of chronic Bright's disease, with the view of relieving the vascular strain by inducing new anastomoses. In some cases the results have been favourable, but it is too soon to express a definite opinion as to this procedure.

XI. PRIMARY CIRRHOSIS OF THE KIDNEY

(GRANULAR CONTRACTED KIDNEY; SMALL RED KIDNEY)

THIS form of cirrhosis of the kidney is in reality a part of arterio-sclerosis, inasmuch as there is invariably a definite change in the arteries generally, similar to what is found in the vessels in the kidney, and associated with this there is hypertrophy of the left ventricle.

Etiology.—There is sometimes a hereditary tendency to arterio-sclerosis, and the great causes of arterio-sclerosis are, and must be, causal factors in the production of cirrhosis of the kidney. These include syphilis, alcoholism, muscular overstrain, overeating, especially of an over-rich diet, gout, and to a less degree, mental overwork. Males suffer more than females, and it is a disease which rarely commences before adult life, while many of its victims suffer at, or after, the age of forty.

A mild form of what is, in reality, arterio-sclerosis, with cirrhosis of the kidney, may be found after death in those who have put too great a strain upon their vessels and kidneys from all sorts of causes, and it is frequently seen in persons who can hardly be termed alcoholic, although they have indulged in an amount of alcohol which has proved excessive for the welfare of their arteries and kidneys. It is an interesting fact that as a result of mental strain arterio-sclerosis is specially prevalent amongst asylum inmates.

A form of cirrhosis may also be the result of hydro-nephrosis, or of any condition where there is similar interference with the renal functions.

Pathological Anatomy.—The kidney is small in size, generally weighing only $1\frac{1}{2}$ to 2 oz.: it is often embedded in fat, sometimes to a remarkable extent, and when this is stripped off, the surface of the kidney presents the granular appearance from which it has derived its name. These granular swellings correspond to the lobules: the capsule is thickened and extremely adherent: the colour is deep red, the cortex, however, being somewhat mottled, if fatty changes are present. The cortex is greatly diminished in thickness, and there are numerous cysts, some of them subcapsular, and others scattered throughout the cortex. They are due to dilated and obstructed tubules: most of them are microscopic in size, but

the larger ones are found to contain a yellowish fluid in which urea and other urinary constituents may be present.

The vessels show an extreme degree of thickening, often resembling endarteritis obliterans, and the vascular markings, although not so irregular as in the "small white kidney," are rarely perfectly straight. Bowman's capsules are greatly thickened and hyaline, and, as a result, the glomerular tufts are often completely atrophied. The interstitial changes are most advanced in the neighbourhood of the interlobular arteries, and therefore the glomeruli farthest away from these vessels are generally less affected than those in their immediate neighbourhood. The tubules have undergone remarkable changes; in parts they are greatly dilated, while the epithelium is often granular and fatty; in some tubules the epithelium is extensively desquamated, and in others it has disappeared altogether. The changes in the heart and arteries have been more fully discussed under the heading of arterio-sclerosis (p. 384).

Clinical Features.—The disease begins insidiously, and may for a long time escape the attention of physician and patient. Amongst the earliest features are tiredness and headache, sleeplessness, dyspepsia, and the passage of an increasing amount of urine, which may necessitate the patient's rising several times during the night.

Urinary System.—The urine may reach 70-150 oz. per day; it is clear and of low specific gravity (1005-1010), with little or no albumin, and contains only a very few hyaline and possibly some granular casts. The urea is generally absolutely diminished. Albuminose are sometimes present in small amount, but their significance is uncertain. Blood is rarely noted unless an acute exacerbation supervenes. Uric acid crystals are sometimes precipitated, probably as a result of the very low pigmentary index and poorness of the urine in salts.

Circulatory System.—The clinical evidences of the arterio-sclerosis are definite; the larger arteries have thickened walls and are often atheromatous, but there is, in addition to actual thickening, a degree of hypertonus, by which is meant vaso-motor spasm, certainly the result of toxins circulating in the blood-stream. The vascular tension may be as high as 150 to well over 200,¹ and the left ventricle is found to be enlarged,

¹ The normal tonometer reading is 80-100; any increase above 100 implies abnormal intravascular pressure.

the apex-beat displaced outwards and downwards, and on auscultation, the first sound in the mitral area is booming in character, and the second sound in the aortic area markedly accentuated. Where enlargement of the left ventricle renders the mitral orifice incompetent, a mitral systolic murmur is heard, and in process of time the evidences of backward pressure appear both in the lungs and the right heart.

These cardio-vascular changes may develop to a quite recognisable extent within six weeks.

Alimentary System.—The appetite is poor, and the long-continued dyspepsia, often associated with morning sickness, soon tells upon the patient's strength; diarrhoea is sometimes present.

Hæmopoietic System.—Anæmia may gradually develop, and a chemical investigation of the blood shows the presence of excess of urea, in cases in which uræmia is impending.

Respiratory System.—Epistaxis is probably the only clinical feature worthy of special reference; it may be both troublesome and excessive.

Integumentary System.—The purpuric hæmorrhages, already referred to, are not uncommonly seen under the skin of the legs, and where, with such hæmorrhages, there is no rheumatic history, the possibility of primary cirrhosis of the kidney should not be forgotten. Pigmentations are also not infrequent in this variety of kidney affection. Dropsy is extremely rare, except in the later stages, or in cases in which an attack of acute nephritis has been superadded.

Nervous System.—The arterio-sclerosis causes very definite cerebral phenomena. Headache, sleeplessness, and difficulty in cerebration are common, and it should be remembered that as many cases of this disease terminate fatally by the development of uræmia, all the uræmic phenomena may be present.

Albuminuric retinitis occurs in a considerable number of cases, sometimes with papillitis and flame-shaped hæmorrhages. Very many cases of apoplexy occur in persons with primary cirrhosis of the kidney, and the prognosis in these cases is invariably more grave owing to the arterial and renal disease.

Diagnosis.—There are no cases which are so difficult to diagnose as early cirrhosis of the kidney, but in the advanced stages the cardio-vascular phenomena and the polyuria render recognition easy. It may, however, be impossible to determine whether the condition has been purely a primary cirrhosis, and

not to some extent a post-inflammatory affection, and, perhaps, the study of the casts in the urine forms the best means of coming to a decision; dropsy is far more frequent in cases which are, or were, inflammatory.

In any case in which a patient comes to the physician complaining of overstrain, languor, and dyspepsia, without any sufficient cause, the possibility of primary cirrhosis should not be overlooked.

Prognosis.—Granular kidney is absolutely incurable, but a long life may be enjoyed by the patient provided the greatest care is taken to guard against strain, the effects of alcoholism, and imprudence as regards exposure to cold and wet.

Treatment.—A quiet life, with little mental and physical effort, must be enjoined upon all patients. Alcohol should be forbidden, and the diet reduced to one which will put little strain on the affected kidneys and vessels. A fairly warm climate and warm clothing are advisable.

Medically we may try to lower the tension where it is excessively high, and probably the nitroglycerine group is the most valuable, because with the relief obtained the headache disappears, and frequently the patient's general well-being much improves. The liquor trinitrini in 1-3 minim doses, or $\frac{1}{100}$ th to $\frac{1}{50}$ th grain of nitroglycerine, should be given. The benefit obtained from potassium iodide in arterio-sclerosis should not be forgotten, and aconite administered in the form of the tincture in 2-5 minim doses often helps to lower the vascular tension. Where the heart is dilated and marked mitral incompetence has developed, digitalis and strophanthus should be ordered, while sometimes venesection or other measures for the relief of backward pressure, and especially the removal of dropsical effusions if they develop, may save the patient at a critical moment.

Purgatives, diaphoretics, and the cautious use of baths, are advantageous in the general treatment of the case.

Anæmia should be treated by the free use of iron.

XII. WAXY DEGENERATION OF THE KIDNEY

WAXY degeneration, especially involving certain vessels in the kidney, is a part of a general condition, and other organs,

PATHOLOGY OF MEDICINE

... in the spleen and alimentary tract,

~~... continued~~ continued suppuration, and bone
~~... amyloid~~ amyloid degeneration, although
~~... with a~~ with a varying degree of
~~... explicable.~~ explicable.

~~... The~~ —The vessels suffer first, and
~~... arterioles~~ arterioles, and also the
~~... period~~ period, the straight arteries and
~~... may~~ may also be affected. The change
~~... coat~~ in the intima coat, and is chiefly limited to
~~... period~~ period the basement membrane
~~... tubules~~ the tubules become involved, and it
~~... epithelium~~ the epithelium may eventually suffer.
The changes associated with the waxy
degeneration is this the case where, in a sub-
acute nephritis, waxy degeneration is super-
added to a syphilitic origin.

The kidneys are in size, weighing 10 oz. to 1 lb.,
and have a glistening appearance; if iodine is
used when the glomeruli are picked out as
specimens. In microscopic sections iodine may
stain the waxy violet, and in both cases the waxy
degeneration stain, mahogany-brown as the
tubules stain rose-pink with methyl-aniline violet.
The waxy degeneration alone, and generally a
tubular, sometimes glomerular, is

~~... The~~ —The urine is large in amount (70-100
oz.) of low specific gravity (1006-1015), and
contains a small amount of albumin; where there
is much albumin there may be much albumin. The
urine is pale and may be described as being of a
pale yellow, although it is doubtful whether they
show any reaction on staining. There are sometimes
granular casts present. The amount of urea
in the urine may be practically normal.

The disease varies greatly; it may be very marked when
anemia and cachexia are present, but it is by no
means a prominent symptom. Cardio-vascular
disease and uremia are rarely present, and where

they occur they result from changes in the kidney other than the waxy degeneration.

A careful investigation should be made so as to determine, *firstly*, the cause of the waxy degeneration; and, *secondly*, the presence of waxy changes in other organs with the respective clinical features.

Diagnosis.—It is obviously impossible by any examination of the urine alone, to recognise positively the presence of waxy degeneration of the kidney, but it may be suspected, where, along with a definite causal disease, the symptoms above mentioned are present.

The **Prognosis** is always unfavourable, and especially when a marked degree of dropsy appears. It naturally depends also to some extent upon the disease to which the condition is due.

The **Treatment** must be upon general lines; it is unwise to give much fatty material, either in the diet or as medicine, and therefore cod-liver oil is contra-indicated where waxy degeneration is present. Potassium iodide, iron, and careful dieting are probably the best methods of attempting to treat a very hopeless condition.

XIII. HYDRONEPHROSIS

DISTENSION of the pelvis and calyces of the kidney with urine, the result of obstruction in some part of the urinary passages.

Etiology.—It is commoner in females. It may be congenital from obstruction of the ureter by folds, imperforate urethra, etc. It may occur in later life from impacted calculus, stricture of the ureter, abdominal and pelvic tumours, growths in the bladder, enlarged prostate, and urethral stricture. In a mild degree it is often associated with floating kidney.

Pathological Anatomy.—The pelvis enlarges at the expense of the kidney; the papillæ of the kidney undergo atrophy, and there may be little kidney substance left. The ureter may participate in the dilatation.

The fluid consists of dilute urine, often with a little albumin; pus is present if there is any inflammation of the pelvis. Where one kidney is alone affected, the other tends to undergo compensatory hypertrophy.

Clinical Features.—Mild cases are often never recognised. If unilateral and more marked it may cause little excepting the evidence of a cystic tumour in the region of one kidney. Not infrequently this tumour disappears with the passage of the contents into the bladder; but generally reappearance soon takes place (the intermittent type).

If both kidneys are involved, in addition to suppression of urine, there is an early development of uramic symptoms.

Rarely does the condition become purulent and form a pyonephrosis.

Diagnosis.—*Ovarian tumours* are generally recognised on vaginal examination, while the hydronephrotic cyst lies in the region of the kidney, and covered anteriorly by the ascending or descending colon. General or loculated *effusions into the peritoneum* may be excluded by examining the fluid after aspiration.

Prognosis.—A unilateral lesion causes little trouble except from the pressure exerted by the tumour, and the condition may even disappear in time without treatment. If the lesion is bilateral and complete, death from uremia must inevitably ensue.

Treatment.—Do not operate on intermittent cases. A pad and bandage have been recommended and may be tried. Aspiration, keeping well behind the colon, is occasionally indicated, while sometimes draining by free incision or even removal of the kidney may be necessary.

XIV. TUBERCULAR DISEASE OF KIDNEY

TUBERCULAR disease may be primary or secondary.

I. *Primary tuberculosis of the kidney* may involve the pelvis of kidney, but also the ureter, bladder, and rarely the prostate, and it is often a matter of difficulty to suggest its starting-point. It generally attacks one kidney first, and if both are involved, one is more markedly affected.

Etiology.—No definite cause for the site of the disease can be assigned. children appear to be less commonly the subjects of this form of tuberculosis.

Pathological Anatomy.—Tubercle nodules are deposited in the substance of the kidney, which apparently start in or

near the pelvis or calyces; these coalesce after caseating, and thus form ragged abscess cavities which open into the pelvis. Lime salts may eventually be deposited in the caseous areas. The ureter is generally thickened, and may be almost occluded with tubercular deposit, and the mucosa may be ulcerated.

Sometimes the bladder, the prostate, the testicles, and the vesiculæ seminales are involved.

Clinical Features.—If only one kidney is affected there may be no symptoms, but usually the case simulates a pyelitis in the early stages, because of the severe lumbar pain; there may be evidence of a renal tumour.

The urine, which is frequently acid, contains pus, and generally tubercle bacilli. Blood is rarely copious, the albumin depends on the pus present, and the amount of urea varies with the capabilities of the unaffected kidney. Later cystitis, with alkaline urine, may develop. Hectic fever with emaciation is present in more rapid cases.

Many cases of extensive renal tuberculosis, exactly similar to primary renal tuberculosis, are really secondary, but they differ in no way from the description given above.

The **Diagnosis** depends mainly on the detection of tubercle bacilli, and evidence of coincident tuberculosis in some other part of the genito-urinary tract, but cystoscopy and testing of the urine from each kidney by the use of Luys' separator may yield invaluable information.

Prognosis.—The condition is compatible with many months or even years of life, but is eventually fatal. Where active tuberculous pyelo-nephritis is present, the immediate outlook is grave.

Treatment.—The possibility of surgical interference should be determined. If the other kidney is healthy, nephrectomy is indicated. Tubercular cystitis contra-indicates operation. Treat otherwise on general lines, cod-liver oil and tonics being useful, while urotropine might be tried as a urinary antiseptic.

II. *Secondary Tuberculosis of Kidney.*—In acute general tuberculosis small tubercle nodules are often found dotted over the kidneys. Sometimes a slight albuminuria is present.

XV. RENAL CALCULUS

(NEPHROLITHIASIS)

THE following calculi are common :—

(1) Uric acid. Hard, round, smooth or finely granular, of reddish colour, generally numerous, poppy-seed to pea in size.

(2) Oxalate of lime (mulberry). Hard, very rough, dark colour, often single.

(3) Sodium urate. Soft, of moderate size.

(4) Mixed calcium and triple phosphates. Phosphates in alkaline urine deposited on a previous calculus (oxalate or uric acid), may be of huge size.

(5) Calcium phosphate. White, smooth, chalk-like, in size from pea to hen's egg.

While calculi are often found in the bladder, they may also occur in the kidney, and especially in the pelvis of the kidney and in its calyces. The most common calculi are uric acid or oxalate of lime, and very frequently carbonate of lime, phosphates, and other salts may be present, forming some of the layers which go to make up the laminated structure of a stone. Cystine and other rare constituents need not be mentioned here; calculi consisting mostly of phosphates generally occur in the bladder, and not in connection with the kidney. The deposition of salts may be the result of some chemical change in the urine, or it may be associated with disease of pelvis or kidney. Most stones have a definite nucleus, the nuclei varying in nature, being sometimes uric acid or oxalate of lime, and sometimes a small blood-clot, or other foreign body. In many cases little uric acid calculi are passed in great numbers, without the definite clinical features which are associated with a large stone, and to this condition the term gravel or sand has been applied.

Etiology.—Calculi occur at all ages; they may be met with in young children, although they are more common after middle life. A good deal depends on the reaction of the urine whether the calculus is on the one hand a uric acid or oxalate of lime calculus, or, on the other, a phosphatic calculus; but it should be remembered that a calculus is always made up of

layers superimposed the one on the other, and in these layers it is very frequent to find evidence of the urine having been from time to time sufficiently alkaline to precipitate carbonate of lime or phosphates.

In children and young persons the uric acid calculus is not uncommon. A urine of high acidity, with a small proportion of salts and a low colour index, tends to precipitate the uric acid normally held in solution. While it is true that concentrated urine may contain an excess of uric acid, it often also contains a sufficient amount of saline constituents to prevent the uric acid from depositing.

In the case of oxalate of lime calculi a chemical error in digestion is probably an important factor, and excess of oxalate of lime will inevitably result in its precipitation from the urine. Green vegetables and fruits of all kinds contain a large quantity of oxalic acid, but certain patients appear to have a sufficient digestive error to explain the oxalate of lime apart from anything specially ingested.

Much stress has been laid upon the association of calculus with a pyelitis, trivial or severe, and it is held to be extremely probable that an excessive amount of mucus may in itself tend to produce the precipitation of certain crystalline bodies from the urine. A sedentary life is a strong predisposing factor in the production of calculus, and assuredly an unsuitable diet, and especially one apt to increase the amount of uric acid in the urine, plays an important part in those cases in which there is at best a tendency to an excessive production of uric acid. It is also true that a diet poor in salts may lead to the precipitation of uric acid. Calculus is much more common in men than in women, the proportion being as 5 to 1, and not infrequently, certainly in one-fifth of all cases, there is evidence of both kidneys being involved.

Pathological Anatomy.—A good deal depends on where the stone or stones are situated. If it is in the pelvis of the kidney, and if, as not infrequently happens, the calculus branches into the calyces, there is a degree of cirrhosis of the kidney induced, and very generally a hydronephrotic distension of the calyces and sometimes the pelvis. In many cases pyelitis is set up, and may even cause the destruction of the whole kidney.

In other cases a calculus may be present for years, and lead to no definite clinical feature which suggests its presence,

or at most a slight pyelitis may be the only pathological result of the stone.

Clinical Features.—Pyelitis, and certainly pus formation in connection with the stone, lead to definite clinical features; but there are many instances of extensive branching calculi which have caused absolute atrophy of the affected kidney, which are only discovered accidentally after death, because the remaining kidney may be adequate for the double duty; small calculi frequently produce no symptoms.

An attack of *Renal Colic* indicates the attempt by the calculus to pass down the ureter. The pain is very typical, it commences in the loin and shoots downwards towards the scrotum, sometimes extending for a considerable distance down the inner side of the thigh. The pain is frightfully severe; it is accompanied by a feeling of sickness, often with vomiting, and the abdominal wall on the affected side is rigid and tender to pressure. The pain frequently occurs in paroxysms, although it may also be constant. The attack lasts for a varying time; it may cease when the stone has reached the bladder, or when it returns to the pelvis of the kidney from which it has been displaced, while in a few cases the calculus becomes encysted and the pain gradually wears itself out. In other cases the pain is located in the loin, and does not appear to shoot downwards towards the leg, and this is more likely to occur where a calculus in the pelvis of the kidney has become displaced, but has not endeavoured to enter the ureter. In both these types of renal colic the temperature is frequently high, 102° or 103° F., while rigors are common, and they may become more marked if pyelitis or suppuration is in progress. Hæmaturia is an almost constant accompaniment of attacks of renal colic, although the amount of blood will depend much on the character of the stone and the irritation which it may set up. For the history and clinical features of stone in the bladder the reader is referred to surgical text-books.

If the stone becomes encysted, the passage of urine from the affected kidney may be arrested and hydronephrosis consequently results. Uremia does not, however, occur unless both kidneys are seriously interfered with.

Diagnosis.—*Biliary* and *intestinal colic* must be differentiated from the colic due to a travelling renal calculus. In biliary colic, the pain is associated with the region of the gall-

bladder, generally with enlargement and local tenderness of that organ, and is very often followed by jaundice, while biliary calculi may appear in the stools after an attack is over. In renal colic the pain shoots, in most cases, towards the inner aspect of the thigh, and there is no special tenderness in connection with the gall-bladder. With enteralgia the pain is frequently better marked in the region of the umbilicus; there is no local tenderness over either kidney, and the history of the attack, commencing with the ingestion of some intestinal irritant, is often definite. Neither in biliary colic nor enteralgia does blood appear in the urine.

In *lumbago* and *neuralgia* the pain may be paroxysmal, but is rarely so severe, and in place of shooting down the thigh would more probably be limited to the affected muscle, or would follow the line of the spinal nerve. There is also in both these affections definite evidence of superficial tenderness on pressure, while blood in the urine is not present.

Blood-clots, associated sometimes with *cancer* of the kidney, may also cause attacks of renal colic. There is commonly in malignant disease much more hæmaturia than with calculus, and sometimes bits of the tumour may be recognisable in the urinary deposit.

In *floating kidney* renal pain is not infrequent, but it does not shoot in the typical direction so often associated with a calculus, and blood in the urine would be exceptional.

It is wise in cases of supposed renal calculus to examine the patient by means of the X-rays, and a photograph often gives more definite and satisfactory information than the use of the screen.

Prognosis.—The patient may only have one attack, but in all probability recurrent seizures imply the formation of other calculi, or the renewed attempt of the original calculus to pass down the ureter. Pyelitis is a common result of renal calculus, while pyonephrosis is by no means an improbable sequel. It is open to question whether the constant irritation of a calculus may not induce the development of malignant disease, and, in any case, there is apt to be a cirrhotic change in the kidney with more or less interference with the renal function. On the other hand, in many cases medical treatment and sometimes surgical interference yield most satisfactory and permanent results.

Treatment.—For an attack of renal colic endeavour to relieve the spasm by hot baths, warm-water enemata, hot fomentations applied over the loins, and hypodermics of morphia (gr. $\frac{1}{8}$ - $\frac{1}{4}$) and atropine (gr. $\frac{1}{100}$ - $\frac{1}{50}$), while in very serious cases chloroform anæsthesia should be employed. Sometimes changing the position of the patient in bed gives prompt relief, and it may help the calculus either to return to the pelvis of the kidney, or to make its way more easily into the bladder; copious draughts of warm water, weak tea, or aerated alkaline water, assist in washing the calculus downwards.

Between the attacks, and after finding out by careful examination of the urine the probable nature of the stone or stones, an attempt may be made to counteract the tendency of the urine to precipitate its constituent salts. In this way alkaline mineral waters, such as Ems, Vichy, Carlsbad, and other spas, have been found to yield good results; while the treatment of the patient with bicarbonate of potash, in 30-40 grain doses, or other alkali, is frequently beneficial. Piperazine has been recommended as a solvent of uric acid; it is given in 15 grain doses, but probably has an extremely feeble effect in the very weak solution in the urine.

Abdominal massage has been commended, but should be used with the greatest caution.

All that need be said here about surgical treatment is simply to urge that the urine from each ureter should be obtained separately, and carefully examined so as to make certain that, if one kidney is operated upon, the other kidney will be capable of taking on double duty, and in certain cases it is probably wise to test the freezing-point of the blood and the urine by the cryoscopic method.¹ Should an abscess be present, surgical interference offers the only chance.

¹ Kummell considers the estimate of the freezing-point of the blood to be of service in renal disease; the larger the quantity of salts and urea the lower is the freezing-point of the blood, while proportionately higher would be the freezing-point of the urine from the inadequate kidney. Normal blood freezes at -56° C., and a fall to -6° C. or lower indicates probable renal inadequacy. Normal urine freezes at 1.2° to 2.3° C. below zero. A rise to 1° or higher indicates a diminution in the amount of salts, and therefore renal inadequacy.

XVI. PYELITIS—PYELO-NEPHRITIS—PYO-NEPHROSIS

INFLAMMATION of the pelvis of the kidney.

Pyelo-nephritis implies that the kidney itself has also become involved, and pyonephrosis that the kidney has broken down and forms an abscess.

Etiology.—Pyelitis may be due to the following:—Calculi; decomposing urine (retained by pressure on ureter) in pelvis, or secondary to cystitis; tubercle, cancer, and parasites; in some of the infectious fevers, such as scarlet, typhoid, small-pox, diphtheria, etc.; and as the result of certain irritating drugs, such as cantharides and turpentine; possibly from cold.

From this list of causes it will be seen that pyelitis may (1) begin in the pelvis of the kidney, as in the case of tubercle; in the infective fevers, where the infective organisms are excreted by the kidney and so enabled to set up a local inflammation; and in any severe illness where it is thought organisms may make their way to the pelvis possibly from the intestine. Pyelitis may (2) be associated with, and be a sequel to, acute nephritis; or it may (3) result from infection from below as in cystitis.

Pathological Anatomy.—There is catarrh sometimes with hæmorrhages, ulceration, and occasionally extensive necrotic areas. In more chronic cases the pelvis and calyces may be distended and the walls greatly thickened. The distension is often sufficient to cause destruction of the kidney substance. Sometimes the kidney suppurates, causing pyonephrosis.

The **Clinical Features** depend on the stage and severity of the condition. Dull, aching pains in the loins, increased on palpation, with mucus and pus in the urine (which may be acid), are common, while the amount of albumin varies. Tailed cells, at one time considered to be diagnostic of pyelitis, cannot be depended on, as they may come from other parts of the urinary tract, and particularly the bladder. In an acute case rigors and hectic phenomena may occur. In chronic cases there is frequently much pus, which is sometimes intermittent from temporary blocking of the affected ureter, in which case a

renal tumour is palpable prior to the discharge of pus. Suppurative nephritis or cystitis may add its characteristic features to those already mentioned.

There is always a risk of uræmia developing.

Diagnosis.—The examination of the urine obtained from each kidney by the urinary separator is an invaluable help, and careful abdominal palpation should be carried out. Tubercle bacilli should be repeatedly sought for.

Prognosis.—Mild cases and those occurring in fevers are favourable. Tubercular cases may become quiescent. Involvement of the kidney, whether it be a pyelo-nephritis or a pyonephrosis, increases the gravity of the case. Prolonged suppuration may cause waxy changes to develop and speedily kill the patient.

Treatment.—Absolute rest in bed, and hot baths, poultices, and sometimes wet-cupping the loins, are advisable in acute cases. Allow plenty of diluent drinks.

Where there is much pus—alum, mineral acids, tincture of the perchloride of iron, and acetate of lead (gr. 3, thrice daily) may be tried, or, with caution, such remedies as oil of turpentine or sandal-wood oil. Urotropine (gr. 10) is sometimes useful, and quinine may be given with benefit. Above all, support the patient's strength by careful but generous diet, and, when possible, send him to the sea-side. In certain unilateral cases nephrectomy or nephrotomy is necessary, but only after carefully testing the adequacy of the unaffected kidney.

XVII. PERINEPHRIC ABSCESS

SUPPURATION around the kidney.

Etiology.—It may be due to (1) A blow or crush; (2) The spread of inflammation from kidney or pelvis outwards; (3) The spread of inflammation from a source outside the kidney, as from perforation of intestine, appendicitic abscess, empyema perforating diaphragm, or caries of spine; and (4) Certain continued fevers in children.

Pathological Anatomy.—The pus may be in different positions; it may have a faecal odour. It may burrow in

different directions, sometimes simulating a psoas abscess, and it may set up peritonitis.

Clinical Features.—A tumour is felt, and pain is commonly present, dull and aching, or sharp and increased on pressure. The leg of the affected side is often kept flexed, adduction is difficult, and the testicle is retracted.

Diagnosis.—Palpation may reveal a definite tumour, and almost always causes local pain. *Hip-joint disease* may have to be excluded. An aspirating needle yields definite information.

The **Prognosis** varies with the nature of each case.

The **Treatment** is surgical, and the abscess should be opened and drained; rarely does the abscess discharge itself spontaneously into the bowel.

XVIII. CYSTS OF THE KIDNEY

1. **Simple Cysts.**—These vary in size from a pea to 3 or 4 inches in diameter, being occasionally even larger. They occur in the cortex, may project from the surface, and contain a clear, watery, or gelatinous fluid, in which there is a trace of albumin and salts, and sometimes urea or uric acid. These cysts are found in kidneys otherwise perfectly healthy, and are by no means of uncommon occurrence. They are probably formed in uriniferous tubules, and are due to the occlusion of these tubules at certain points. They are very rarely palpable, and are generally discovered after death. It has been suggested that possibly injury might explain the formation of some of these cysts, but there is little evidence in favour of this view.

2. **Cysts** occur also in *granular contracted kidney*, and are specially common in primary cirrhosis, although they may be present in any of the chronic forms of Bright's disease. They are of small size; sometimes they contain fluid dark in colour from the presence of blood pigment, and they cause no symptoms.

3. **Dermoid cysts** may occur in the kidney, although they are exceptionally rare.

4. **Hydatid cysts** are also found, to which reference is made under the head of parasites of the kidney (p. 532), and also parasites generally (p. 184).

5. **Cystic Diseases of the Kidney.**—This condition may occur in adults, or it may be congenital. The whole organ is composed of cysts varying in size, containing a clear or opaque, sometimes viscid fluid, which may be reddish in colour, occasionally blackish. Albumin, blood, leucocytes, and cholesterin have been found in the fluid. All distinction between the cortex and the medulla of the kidney becomes lost, and it is difficult to detect any normal kidney substance in an advanced case. The kidneys are usually enlarged, and sometimes very greatly, and the tumours formed by them may be readily palpable. The condition is bilateral in the form occurring in adults, and the enlargement in that form reaches a greater size than in the congenital variety.

Many theories have been suggested to explain cystic disease. Some authorities hold that the cysts are formed by obstruction of tubules, others that persistence of the Wolffian body, which has become mixed up with the kidney, may explain the condition, but in quite a number of cases the cystic disease is not limited to the kidneys only, the liver being often affected, while cysts have been noted in the brain in patients who have well-marked cystic disease of the kidneys. It seems probable, therefore, that cystic disease may be the result of a new growth in which cysts form, but this theory is open to question.

Clinical Features. Many cases are only discovered after death, although death may be the result of uræmia with fairly typical symptoms. The clinical features, when present are practically those of uræmic poisoning, and include nausea and vomiting, debility, ill-health, anæmia, and very generally the passage of a large quantity of pale urine of low specific gravity, and suggestive of cirrhotic kidneys. There may be a trace of albumin, but dropsy is certainly uncommon; other uræmic phenomena, such as dyspnoea, twitchings and cramps, and coma, together with pyrexia, may develop towards the termination of the disease. The physical signs would be distinct were the abdomen palpated in suspicious cases, because in cystic disease in adults the kidneys are nearly always sufficiently large to permit of easy recognition. The cardiovascular changes associated with cirrhosis of the kidney are not infrequently present in cystic disease.

Diagnosis.—As already noted, cystic disease is often

missed entirely, but the presence of renal tumours, with the clinical features of cirrhosis of the kidneys, should make the diagnosis fairly clear.

The **Prognosis** depends on the adequacy of the kidneys for their vital functions, and also upon the amount of strain which the patient puts upon them. Any evidence of uræmia is of grave omen.

Treatment.—The symptoms may be combated, but otherwise little can be done. It has been suggested that a kidney might be removed, provided one only is involved, but there seems no particular object to be gained by this procedure.

XIX. EMBOLISM OF THE KIDNEY

THIS is a common result of ulcerative endocarditis, but may occur where small clots, not necessarily containing pyogenic or other organisms, are broken off from aortic or mitral vegetations, or from calcareous plates on atheromatous arteries. The emboli are often multiple, and in cases of pyæmic nature may be found in the capillaries. The blood-supply of the affected area of kidney is arrested, and fatty changes ensue, and where suppurative organisms are present, acute necrosis, sometimes even gangrene, may result. In simple cases (where there are no organisms) the infarct gradually becomes absorbed, and is partially replaced by fibrous tissue, leaving a scar easily seen on the surface of the kidney. Infarctions are usually limited to the cortical portion of the organ.

Clinical Features.—There may be no symptoms at all, or the passage of an embolus to the kidney, with resulting infarction, may give rise to sudden local pain, and to the presence of albumin, largely due to the appearance of blood in the urine, and in simple cases these symptoms may last for one or two days. If organisms are present, rigors often ensue, and the pain is usually more severe. In pyæmic cases rapid death, mainly due to the general condition of the patient, is inevitable.

The **Diagnosis** is usually fairly easy where sudden pain and blood in the urine are present, together with evidences of valvular disease. In the absence of pain and blood in the urine a simple infarction cannot be diagnosed.

The **Prognosis** depends on the presence or absence of organisms, and on their nature.

The **Treatment** is purely palliative.

XX. PARASITES OF THE KIDNEY

THE more important parasites have been referred to in a separate section (p. 182), but a further reference is desirable to several of these.

1. **Hydatid Cysts**.—The cysts are generally found in the substance of the kidney; they may be under the capsule, or they may destroy the whole of one organ. They tend to discharge into the pelvis of the kidney, and may produce severe attacks of renal colic from the vesicles which attempt to pass down the ureter. A large hydatid cyst may form a considerable abdominal tumour, readily palpable, although it is unlikely that the hydatid thrill, always of doubtful existence, can be definitely made out.

The **Diagnosis** frequently depends on aspiration, and the clear watery fluid, of a specific gravity of 1005-1009, containing common salt, but no albumin, and often with hooklets, and even scolices, is distinctive.

The **Treatment** consists in an attempt to empty the cyst, sometimes by aspiration, and sometimes by stitching it to the abdominal wall. A cyst may fill up after aspiration, or a neighbouring cyst may take the opportunity of growing rapidly. Unfortunately, hydatid cysts, if present in the kidney, are apt also to be in the peritoneum and elsewhere, and therefore the treatment is not invariably successful. If the cyst ruptures into the pelvis of the kidney, large quantities of fluid should be drunk by the patient, in order to aid the expulsion of daughter cysts by the ureter, and warm baths, together with the other treatment for renal colic, should be carried out.

2. **Bilharzia Hæmatobia**.—This parasite is described on p. 192; it is a cause of hæmaturia, and generally the ova are passed from time to time, and, together with the ciliated embryos, may be readily recognised under the microscope. The ova may cause a tedious illness, from which, however, the patient generally recovers, and the treatment consists in

alkaline drinks and in doses of oil of turpentine (℥ 15-20), or of filix-mas (℥ 15-60).

3. **Filaria Sanguinis Hominis** is described on p. 189 ; it causes chyluria and hæmaturia, and may be associated with the development of elephantiasis.

4. **Eustrongylus Gigas**.—This is an enormous nematode, the female being 3 feet in length and the male 1 foot, of very rare occurrence in man. When it is present in the kidney, it causes complete disintegration of the organ.

5. **Pentastoma Denticulatum**.—The larval form of the *Pentastoma tænioides*, the latter being small worms a few inches in length, found in the frontal sinuses and nostrils of dogs, and very rarely in man. The larval form may occur in the kidney without apparently causing very much interference either with its structure or functions.

XXI. TUMOURS OF THE KIDNEY

1. **Simple Tumours** are by no means uncommon in the kidney. They may be small fibromata frequently seen near the apices of the pyramids, also lipomata and angiomas, while occasionally a suprarenal gland is enclosed inside the kidney substance. None of these tumours cause any symptoms as a rule.

2. **Malignant Tumours**.—(1) *Sarcoma*.—*Primary sarcoma* is more common in children ; it may grow to an enormous size, and sometimes a rhabdomyoma occurs which shows a remarkable similarity to striped muscle. Sarcomatous tumours may reach many pounds in weight—twelve pounds, even to thirty pounds—and may completely fill the whole abdomen. They grow rapidly and cause hæmaturia, a varying amount of pain sometimes due to the passage of clots down the ureter, and the development of a recognisable tumour on palpation.

Secondary sarcoma may infiltrate both kidneys, but does not give rise to such definite phenomena as a primary tumour.

(2) *Carcinoma*.—Cancers may be *primary* or *secondary* ; the *primary* form is comparatively rare, but the tumour, when it does occur, may attain a great size, while *secondary* tumours cause comparatively slight symptoms. Cancers may be encephaloid and sometimes colloid.

Diagnosis.—Palpation usually affords very definite information; the tumour growing from the region of the kidney is usually readily distinguished from an *enlarged spleen*, or from *sarcomatous* involvement of the *retro-peritoneal glands*. In certain cases it may be difficult to make out a renal tumour from a *tumour* of the *liver*, but the liver tumour moves more freely downwards with the diaphragm, while a renal tumour is more definitely moored, and is not so readily displaced when the diaphragm contracts. The presence of blood in the urine, and blood often in great excess, and the passage of clots accompanied by colicky pain, generally render the diagnosis easy.

The **Prognosis** of malignant tumours is invariably serious: the duration of life depends, however, partly on the possibility of the affected kidney or kidneys carrying on the functions of nature, and partly on the exhaustion and emaciation which every malignant growth naturally causes. In certain cases renal arteries and veins are invaded by the tumour growth, and in this way a fatal result may be hastened.

The **Treatment** must be purely on palliative lines, the pain generally requiring sedatives and rest in bed. The application of ice locally, and administration of astringents internally, are often of value for hæmaturia. Order soothing liniments, and the use of belladonna or aconite internally, and, where absolutely necessary, opium, to alleviate the patient's suffering.

Surgical treatment is advisable in some cases, certainly only in primary tumours, and where there is a reasonable hope of the tumour being localised to the kidney, and to the kidney alone.

DISEASES OF THE NERVOUS SYSTEM

I. INTRODUCTORY

Neurone.—The Nervous System is made up of neurones, and the conception of a neurone has been much modified within recent years by numerous researches. It consists of a cell-body made up of a nucleus containing a nucleolus, and surrounded by protoplasm in which are granules, commonly called Nissl's granules, which vary in arrangement and appearance in different types of cells. There are grey branching processes or dendrites, which, for a certain distance from the cell-body, contain Nissl's granules, and there is usually one axis-cylinder process, neurite or axone which has a specialised structure. The axis-cylinder processes give off collaterals at different levels. In a peripheral nerve such a process consists of the axis-cylinder surrounded by a sheath of myelin, which in turn is surrounded by a sheath termed the neurilemma sheath. This neurilemma sheath consists of cells, epithelial in character, each of which surrounds the portion of axis-cylinder between two nodes of Ranvier. The axis-cylinder terminates in muscle or skin, or, where it passes upwards or downwards in the central nervous system, it may pass to the cell or cells of other neurones. The axis-cylinder is made up of numerous neuro-fibrillæ, many of which run through the cell to enter one of the grey processes, while fibrillæ, which are also present in the grey processes, may pass from one grey process into another without being interrupted in the cell-body.¹ It has therefore been thought that stimuli may not originate in the cell-body itself, but possibly in the terminal portions of the grey processes, because it seems certain that an impulse can pass along these neuro-fibrillæ without being interrupted in the cell.

¹ Some authorities deny this, and consider that the neuro-fibrillæ are artificial.

Some cells, such as the cells of the posterior nerve-root ganglia, have a single axis-cylinder passing from them, which divides into two parts, one part passing downwards into the nerve and the other up into the cord.

Much discussion has arisen in connection with the directness of the communication between one neurone and another, some authorities asserting that the neurones are distinct, and others that the axis-cylinder or the grey processes of one neurone may anastomose with the grey processes of another; but whether direct continuity has been proved or not, there is direct transmission of impulses from one neurone to another.

Waller's law is as follows:—If an axis-cylinder process is separated from its trophic cell-body, it degenerates, and it has been proved that the cell-body of each neurone is, to a certain extent, trophic for all its processes.

It has also been shown that, if a process be divided, the cell-body suffers,—the *réaction à distance*,—and it may be stated briefly that it suffers in the following way:—

(1) The cell initially swells up, although eventually it atrophies.

(2) The grey processes diminish in number.

(3) Nissl's granules undergo marked alteration, becoming less numerous and being diffused throughout the protoplasm of the cell, a condition termed *chromatolysis*.

(4) The nucleus, which often becomes somewhat diminished in size, tends to assume an eccentric position, and has even been found extruded entirely from the cell.

Most of the cells recover after such a lesion. Toxins acting on neurones produce very similar changes in their cell centres, but the certainty of recovery depends on the nature of the toxin in question.

It is impossible to discuss the mode of regeneration of a divided axis-cylinder. It is sufficient to say that, whereas the theory that it develops from the cell outwards is unquestionably correct, there is reason to suppose that this process is aided by peripheral regeneration, and that the neurilemma nuclei may take on the function of neuroblasts.

It is unnecessary to describe the terminations of sensory and motor nerves, and all that can be attempted is to offer a brief and elementary account, accompanied by diagrams, of the course of fibres, motor and sensory, in the brain and in the spinal cord.

MOTOR FIBRES

The motor neurones may be divided into two groups—those belonging to the First or Upper Motor Neurone or Trophic Realm, and those belonging to the Second or Lower Motor Neurone or Trophic Realm.

I. The First or Upper Motor Neurone, aptly called by Professor Wyllie the First Trophic Realm, extends from the cell centre in the motor cerebral cortex, through the internal capsule, and down into the cord either by the crossed pyramidal tract, in which case the fibre has decussated at the decussation of the pyramids in the medulla, or by the direct pyramidal tract, passing down the same side of the cord and decussating in the cord itself. This first motor neurone terminates in the neighbourhood of grey processes of one or more cells in the anterior cornu of the spinal cord; whether it comes into actual contact or not is not yet proved.

II. The Second or Lower Motor Neurone, or the Second Trophic Realm, has its cell centre in the anterior cornu of the cord, and the fibre passes through the anterior nerve root into a motor nerve.

In a lesion of the First or Upper Motor Neurone, if it be irritative and involve the cortical *centre*, Jacksonian epilepsy results, but if it is destructive, it may cause complete paralysis. A lesion of the *axis-cylinder* of this motor neurone causes interference with voluntary control over the reflex arcs, and therefore exaggeration of tendon reflexes will be found present, and will follow in most cases after a limited time, and when the patient has recovered from the shock of the injury. Only in very severe and sudden lesions is it found that this over-excitability of the reflex arcs is abolished for ever. A lesion of the fibres of this motor neurone may cause complete paralysis, but there is almost always a considerable amount of spasm, with a tendency to contracture. The muscles do not waste except from disuse, and there are no qualitative changes in the electrical reactions.

In a lesion of the Second or Lower Motor Neurone the lesion must either be situated in the anterior horn of the cord, as in infantile paralysis, or else it must involve the anterior nerve roots, or the motor fibres in the nerves as in peripheral neuritis. The cells in the anterior horn of the cord are trophic

cells for the muscles which the nerve supplies, and therefore the muscles waste and the electrical reactions become greatly changed.

The Reaction of Degeneration.—The degenerated nerve is not stimulated by either the galvanic or faradic current, and the muscles cease to react to the faradic current after a very short time. Normally with the galvanic current applied to the muscle, on closing the circuit, the negative pole or cathode gives the first and strongest contraction, the positive pole or anode being distinctly weaker. This is expressed by the formula C.Cl.C is > An.Cl.C. In the reaction of degeneration there is (1) a polar change, the anodal closing contraction being more powerful than the cathodal, so that An.Cl.C is > C.Cl.C. With regard to the opening contractions, it is usually stated that the anodal opening contraction is stronger than the cathodal, and that in the reaction of degeneration this is reversed. In addition to the polar change (2) a weaker current causes contraction, and (3) the contraction, when obtained, is *much more sluggish* than it is in health. These phenomena constitute the reaction of degeneration, and they generally develop about seven to ten days after a lesion of the nerve supplying the muscle.

An *Incomplete Reaction of Degeneration* implies that the conducting capabilities of the nerve are not entirely destroyed. The faradic and galvanic excitability of the nerve may be only lessened, while in place of definite polar change, on testing the muscle, the closing contractions with cathode and anode may show equal excitability. Certainly the retention of faradic irritability is most important, and generally indicates that a rapid recovery may be anticipated.

The multipolar cells of the anterior horn of the cord are also centres of reflex action both of skin or superficial reflexes and tendon or deep reflexes, while in the lumbar enlargement of the cord certain of these nerve cells constitute the motor centres for the great organic reflexes of bladder and bowels. The superficial and deep reflex arcs are under the control of centres in the brain, and the tracts by which these controlling impulses pass downwards are the pyramidal tracts. A lesion of these pyramidal tracts generally causes spastic paralysis with exaggerated knee-jerks, because inhibitory stimuli from the brain are interfered with. On the other hand, a lesion of the cells in the anterior horn of the cord, damaging the great

trophic centres for muscles and also the motor centres of the reflex arcs, causes abolition of both superficial and deep reflexes ; if the lesion involves the lumbar enlargement, the organic reflexes of bladder and bowels also suffer.

Myotatic Irritability.—It was formerly supposed that upon tapping a tendon such as that of the quadriceps extensor muscle a message was sent by way of sensory fibres to cells in the anterior horn of the cord, from which in turn a stimulus travelled by the sciatic nerve to the muscle, causing contraction. It has been proved that the time taken for the passage of these nerve stimuli exceeds in length the period between the tap on the patellar tendon and the eliciting of the resultant knee-jerk. Gowers therefore suggested that the muscles might be kept in a state of tonus by the integrity of the reflex arc, and that it was not necessary for a message to travel upwards to the cord and downwards again to the muscle in order to bring about muscular contraction, but that the muscle, being in a state of tonus or myotatic irritability, contracted directly upon the tapping of its tendon.

It is important to remember the segments of the cord to which the various superficial and deep reflex arcs belong, and in the short tables appended, reference is made to the mode of eliciting these reflexes.

Table of Superficial Reflexes

| | | | |
|----|--------------------|--------------------------------------|--|
| 1. | Scapular reflex. | 5th cervical to 1st dorsal segments. | Is elicited by drawing the finger over the scapula, causing contraction of the scapular muscles. |
| 2. | Epigastric reflex. | 4th dorsal to 7th dorsal segments. | Is elicited by drawing the finger across the hypochondriac region when the epigastrium is indrawn. |
| 3. | Abdominal reflex. | 9th dorsal to 12th dorsal segments. | Is elicited by drawing the finger down the lateral aspect of the abdominal wall, causing contraction of the abdomen. |

Table of Superficial Reflexes—continued

| | | | |
|----|---------------------|------------------------------------|---|
| 4. | Cremasteric reflex. | 1st lumbar to 2nd lumbar segments. | Is elicited by tickling the inner aspect of the thigh, causing contraction of the cremaster muscle with drawing up of the testicle. |
| 5. | Gluteal reflex. | 4th lumbar to 5th lumbar segments. | Is elicited by drawing the finger across the buttock, causing contraction of the gluteal muscles. |
| 6. | Plantar reflex. | 1st sacral to 2nd sacral segments. | Is elicited by tickling the sole of the foot, which causes flexion of the toes. |

Table of Deep or Tendon Reflexes¹

| | | | |
|----|--|--|---|
| 1. | Supinator longus and biceps jerks. | 5th cervical to 1st dorsal segments. | Are elicited by tapping the tendons of these muscles. |
| 2. | Triceps-jerks. | 6th cervical to 7th cervical segments. | Is elicited by tapping the triceps tendon with the arm flexed. |
| 3. | Knee-jerk. | 2nd lumbar to 4th lumbar segments. | Is elicited by tapping the patellar tendon, causing extension of the leg at the knee-joint. |
| 4. | Achilles jerk. | 1st sacral to 2nd sacral segments. | Is elicited by tapping the Achilles tendon, causing contraction of the calf muscles. |
| | Ankle-clonus or rhythmic contraction and relaxation of calf muscles. | 1st sacral to 2nd sacral segments. | Is elicited by suddenly putting these muscles on the stretch. |

¹ The following reflexes are of less importance, but might be added :—(1) Adductor-jerk is obtained by abducting the thigh and tapping the adductor magnus tendon. A crossed adductor-jerk has been sometimes noted. (2) Jaw-jerk is elicited by making the patient open the mouth, placing one finger on the chin and tapping sharply on it, when the jaw muscles contract, and the head is jerked forward. It is absent normally.

SENSORY FIBRES

The Sensory Fibres are much more complicated than the motor. Not merely is it probable that there are a considerable number of different fibres subserving different kinds of sensation, but in addition, when these fibres are traced upwards towards the brain, they become extremely involved in their arrangement. According to recent researches by Head there are three sets of sensory fibres carrying different sensations.

(1) There are fibres which subserve deep sensibility, and conduct impulses produced by movement. These fibres run, Head believes, in motor-nerve trunks, and are not destroyed by the division of all the sensory nerves to the skin.

(2) There are fibres which subserve what Head calls *protopathic* sensibility. Such fibres respond to painful cutaneous stimuli and to the extremes of heat and cold, and they also endow the hairs with the power of reacting to painful stimuli. They regenerate rapidly when the ends of a divided nerve have been reunited, the conductivity of these sensory impressions returning in 7 to 10 weeks.

(3) There are fibres which subserve *epicrytic* sensibility. These fibres carry stimuli due to the lightest touch, and enable the individual to recognise the finer grades of temperature. They regenerate more slowly, and after suture of a divided nerve do not regain their normal functions until 6 months at least have elapsed.

The sensory nerve fibres can only be referred to in an elementary way, which, however, is sufficient for practical purposes. There are systems of sensory neurones which connect with each other and carry stimuli upwards, and there are at least three of these between a sensory end organ and the cerebral cortex.

The First or lowest system is represented by the neurones, whose cell centres are in the posterior nerve-root ganglia. These cells have a single axis-cylinder process which divides close to the cell—one axone entering the nerve, and the other passing up into the cord. The axone in the nerve corresponds to and is the only representation of the dendritic processes usually possessed by nerve cells, and, like dendritic processes, it conducts stimuli *towards* the cell. The axone which enters the spinal cord divides into an ascending and a descending

branch. The descending branches give off collaterals, which terminate like themselves near cells in the grey matter of the cord. The ascending branches of the axones, or the collaterals given off from them, are distributed as follow:—(1) They may run upwards to the medulla in the postero-internal (from lower nerve roots) and the postero-external (from upper nerve roots) tracts, terminating near the cells of the nucleus gracilis and nucleus cuneatus respectively. These fibres do not decussate. (2) They may pass out into the postero-external tract, then bending backwards, may re-enter the posterior horn, and terminate near the multipolar cells of the anterior horn, forming the sensory limbs of the reflex arcs. (3) They may terminate near cells of Clarke's vesicular column, from which the direct cerebellar tract originates. (4) Many fibres terminate near cells in the grey matter of the posterior horn, whose axones form the antero-lateral tract of Gowers, while fibres carrying pain, temperature, and tactile impressions also terminate near cells in the grey matter. Lastly, there are many axones which decussate, but whose destination is uncertain.

The Second System contains the following sensory neurones:—(1) The cells of the nucleus gracilis and nucleus cuneatus send their axones partly by the fillet of the same and partly by that of the opposite side to terminate in the thalamic region, or else to form the columno-cerebellar tract which goes to the lateral lobe of the cerebellum. (2) The cells in Clarke's vesicular column send their axones to the direct cerebellar tract, mostly on the same side. These axones pass through the restiform body to terminate in the middle lobe of the cerebellum. (3) Cells widely scattered throughout the grey matter of the posterior horn send their axones to the opposite antero-lateral tract of Gowers and to neighbouring nerve bundles. Gowers' tract ends probably in the middle lobe of the cerebellum.

The Third System of sensory neurones is much too intricate to receive adequate description here. In part the cells are in the thalamic region, and their axones pass to the cortex behind the Rolandic fissure. There are also many commissural neurones belonging to this system which are extremely complicated.

If the reader glances at the description given of Brown-

Séguard's paralysis, he will note that the fibres carrying common sensation, tactile or finer sensation, and temperature sense, decussate soon after entering the cord, while fibres conducting muscular sense pass up on the same side. It seems probable that of the fibres which decussate, those subserving common sensation and temperature sense pass upwards near the grey matter and not far from Gowers' tract, while the muscular sense fibres must be situated in the posterior columns. The path of the tactile sense fibres is still unknown.

II. DISEASES OF NERVES

1. LOCAL NEURITIS

Inflammation may arise in various structures of a nerve.

(1) In the outer fibrous sheath surrounding the funiculi, called *perineuritis*.

(2) In the delicate connective-tissue fibres dividing up the constituent nerve elements in the funiculi, called *interstitial neuritis*; or

(3) An inflammation or degenerative change actually involving the nerve fibres themselves, called *parenchymatous neuritis*.

The term interstitial neuritis is generally used to include perineuritis.

It is rare to find parenchymatous neuritis alone, without a certain amount of interstitial change. Parenchymatous neuritis is best exemplified by multiple or peripheral neuritis, generally of toxic origin, and described separately.

Etiology.—A localised perineuritis commonly results from rheumatism, gout, or exposure to cold. A nerve may also be injured by wounds or bruises, fractures, tumours, abscesses, and so forth, and in these cases a perineuritis is, at all events, first produced, although later the funiculi may be involved. Toxic neuritis is referred to separately under multiple neuritis, but where a nerve is affected by some local condition, which alone might be insufficient to produce a neuritis, a toxin capable of acting on the peripheral nerves may specially devote its attention to those nerves which are below par. And in the same connection it may be stated that, in alcoholic neuritis,

exposure of one or more limbs to great cold may specially predispose to an attack on the nerves of these limbs.

Pathological Anatomy.—In perineuritis and interstitial neuritis there is generally an exudation into the connective tissue; sometimes it is of inflammatory lymph, occasionally there is an invasion of leucocytes, and in very acute cases hæmorrhages are not infrequent.

Inside the funiculi similar interstitial changes may be found. The parenchymatous change in the nerve fibres is described under multiple neuritis.

Clinical Features.—In some cases there is a marked degree of constitutional disturbance, but locally the chief feature is pain, and pain shooting down the line of the nerve. It increases with pressure over the nerve, and is generally more severe at night. The skin may be hyperæsthetic, and later in the affection, anæsthetic. Sometimes it is possible to make out on digital examination that the nerve is definitely swollen. Should the nerve carry motor fibres, the muscles become weakened and eventually paralysed, and muscular tenderness and sometimes fibrillary twitching may be noted.

The trophic changes vary considerably; the skin may be reddened and swollen, sometimes it is thin and glossy; the nails may become brittle and even fall off. Local sweating, herpetic eruptions, joint swellings, and other trophic changes are not uncommon.

Diagnosis.—In most cases the diagnosis is easy, although the exact nature of the etiological factor may not be clear. The possibility of rheumatism and gout, as well as syphilis, and, of course, exposure to cold, which is the most frequent cause, should be carefully investigated.

The **Prognosis** varies considerably. In some cases benefit rapidly ensues from rest and treatment. In other cases where there has been much exudation, a long period elapses before the nerve can resume its normal functions.

Treatment.—Rheumatism, gout, and syphilis should be appropriately treated; rest must be ordered for the affected part, and warm applications applied along the line of the nerve. The pain may be so excessive as to demand the use of opium or morphia, and sometimes local counter-irritation (provided no trophic change is in evidence) may be applied—such remedies as iodine and fly-blisters being of great service.

Potassium iodide is invaluable, and the constant current not merely relieves pain, but also helps to remove exudation. In some cases an attempt may be made to puncture the nerve by the method of acupuncture referred to under sciatica.

2. NEURITIS MIGRANS

Is a remarkable form of neuritis in which the inflammatory process generally travels up the nerve, causing intense pain, sometimes so excruciating as to determine the patient in favour of amputation. It is generally due to a septic wound, or, at all events, a wound of the nerve peripheral to the ascending inflammation, and the presence of an irritant of some kind is essential.

In these cases the best treatment is to search at the site of the injury for a neuroma, or other possible source of irritation, or to divide the nerve; in not a few instances treatment fails, and amputation of the limb affected has to be performed.

3. SYMPATHETIC NEURITIS

Implies inflammation of the corresponding nerve on the opposite side of the body, secondary to the primary involvement of a nerve by an abscess, tumour, or other lesion.

4. MULTIPLE NEURITIS OR PERIPHERAL NEURITIS

This is a symmetrical and peripheral neuritis, the lesion being best marked near the peripheral end of the nerves. Although probably in most cases the whole neurones suffer, the first clinical evidences of involvement are found peripherally. Nearly all the forms are true parenchymatous neuritis.

Etiology.—The following grouping of causes will be found helpful: (1) Diffusible stimulants, as alcohol, naphtha, bisulphide of carbon, dinitro-benzine, etc.; (2) Metallic poisons, as arsenic, lead, mercury; (3) Toxins from the micro-organisms of diphtheria, typhoid, rheumatism, septicæmia, syphilis, pneumonia, tubercle, malaria, beri-beri, leprosy, etc.; (4) It may also occur in diabetes mellitus, possibly in gout, in cachexia, cancer, and sometimes in profound anæmia.

Cold has been described as a cause of the condition; probably it is an important exciting cause where alcohol or other predisposing factor is present. Similarly, physical ill-health or mental depression has a potent influence. In the above groups certain of the lesions are primarily rather an interstitial than a parenchymatous neuritis.

Pathological Anatomy.—The myelin and the axis-cylinders of the affected nerve fibres break up, the neurilemma nuclei proliferate, and there are almost invariably two associated conditions seen in fatal cases, namely, a marked degree of thickening of the coats of the arteries and capillaries in the nerve, and exudation inside the funiculi, sometimes hæmorrhagic, sometimes leucocytic, and sometimes mostly inflammatory lymph. The cell centres belonging to the affected neurones show chromatolysis, eccentric position, and sometimes even extrusion, of nuclei.

In the muscles there are also marked secondary changes, including loss of striation, fatty degeneration, and proliferation of nuclei. In cases of sufficiently long standing there is, we believe, evidence of peripheral regeneration of nerve fibres in the affected nerves.

Clinical Features.—As a type it is best to take alcoholic neuritis. This often occurs in women, and is common in drinkers of all classes, but coexisting ill-health is a frequent and most important predisposing cause. Many of these cases are believed by some authorities to be really due to contamination of beer or other alcoholic liquor with arsenic, and to be cases of arsenical neuritis.

There are different types—such as sensory, motor, etc.—indicating a predilection of the toxin for certain neurones, and the condition may be acute, subacute, or chronic. The early *Sensory* symptoms are pain, tingling, and numbness, and cramps and pains on pressure over nerves and muscles; the chief *Motor* symptom is paresis of the extensors of hands and feet causing wrist-drop and foot-drop.

The *Sensory Phenomena* are, besides the symptoms mentioned above, delay in conduction of sensations, the patient often crying out a few seconds after the muscle or nerve has been pressed, and sometimes alteration of the temperature sense.

The *Motor Phenomena* are, besides wrist-drop and foot-

drop, high-steppage gait, weakness of muscles of back and neck, and tremor or rather tremulousness of the limbs. Death may be due to involvement of the phrenics and the intercostal nerves, sometimes the vagi. In the leg the extensors, tibialis anticus, and peronei suffer specially and generally first of all. Inco-ordination is a marked feature in certain cases.

The *Reflexes*.—The knee-jerk and wrist-jerk are usually lost, but are sometimes exaggerated. The superficial reflexes soon disappear, even if exaggerated at first. The organic reflexes are generally unaffected, except in very severe cases or as a late phenomenon of grave import.

The *Electrical Reactions* are diminution to faradic stimulation, and the reaction of degeneration in the affected muscles (see page 538).

The *Vasomotor Phenomena* include pallor, redness of hands and feet, cedema of ankles, and a profuse and generally ill-smelling perspiration.

Trophic Functions.—The affected muscles waste, the affected skin may become glossy, the nails brittle, and bedsores are to be dreaded in severe cases.

The *Special Senses* are usually normal.

Mental Condition.—There is great loss of memory, and hallucinations, often very vivid, are almost constantly present.

Cardiac weakness is a serious complication, and the lungs must be regularly examined, because a low type of pneumonia may occur. The temperature varies; in acute cases it may be high, 102°-103° F., in more chronic cases it may never be above normal.

The duration is uncertain; in an acute case death may occur from involvement of respiratory muscles, vagi, or heart in 10-14 days, while a chronic case may take weeks to reach its climax, and months before complete recovery is attained. Pain and tenderness disappear first, and sensation returns long before motor power.

Diagnosis.—The symmetrical distribution and the pain on pressure over nerves and muscles usually render the diagnosis clear, because in *infantile paralysis*, *locomotor ataxia*, *Landry's paralysis* and *hysteria*, these phenomena do not correspond. On the other hand, in very acute cases the disease may be almost indistinguishable from Landry's paralysis.

Prognosis.—Very acute cases are generally fatal. Pul-

monary and other complications must be guarded against and their appearance is serious. Chronic cases are favourable, but may be extremely slow. Returning faradic irritability is a sure sign of restoration of function.

Treatment.—Remove the cause, absolutely confine the patient to bed for a time, and feed carefully.

Hot fomentations are most helpful, and sometimes hypodermics of cocaine or even morphia. Bromides and chloral may be given to soothe the mental excitement, and as soon as possible, *i.e.* when the inflammation in the nerve is arrested begin massage, daily hypodermics of strychnine (gr. $\frac{1}{6}$) and electricity, both faradic and galvanic. Sand-bags are of value where there is foot-drop, to correct any tendency to contracture.

2. ARSENICAL NEURITIS.—(See Arsenical Poisoning, p. 175.)

The clinical features closely correspond to the alcoholic type, the legs suffering more than the arms. There may be inco-ordination, and there is very marked tremor.

3. LEAD PARALYSIS.—(See Lead Poisoning, p. 174.)

The following types of paralysis occur, and the table appended may be useful for reference:—

(1) Antebrachial type, in which there is paralysis of the extensors of the fingers and the wrist; but the supinator longus and extensor of thumb escape. The typical wrist-drop results.

(2) Brachial type, including paralysis of the deltoid, biceps, brachialis anticus, and supinator longus, but the pectoral muscles escape.

(3) Duchenne-Aran type, in which the small hand muscles are affected, as in progressive muscular atrophy.

(4) Peroneal type, in which the peronei, the extensor longus digitorum, and the extensor of the great toe are affected, giving foot-drop.

(5) Laryngeal type, in which the adductors suffer.

Remember that the arms suffer most frequently and that the supinator longus and the extensor muscles of the thumb usually escape.

5. TUMOURS OF NERVES

NEUROMATA.—These tumours either consist of nerve fibres sometimes even of nerve cells, in which case they are called

“true neuromata,” or else they are composed of connective tissue constituting the far more frequent “false neuromata.” True neuromata usually consist of nerve fibres, and the suggestion has been made that where ganglion cells are present, it is the result of a congenital malformation. Gliomatous tumours closely resemble true neuromata in appearance, although microscopically they are easily distinguished.

1. **MULTIPLE NEUROMATA** are tumours intermediate between false and true neuromata. They occur on many of the nerves of the body, and occasionally give rise to considerable pain when the tumours are palpated.

2. **MULTIPLE NEUROFIBROMATA.**—These growths, described by, and called after, von Recklinghausen, are amongst the most remarkable tumours in existence. The essential features are :—

(1) Numberless small fibrous nodules, some sessile, others pediculated, which are situated in the skin. These vary in size, and may be extremely large.

(2) Tumours on the nerve trunks, very similar to those described under multiple neuromata, and they cause considerable pain, cramps, and even paralysis.

(3) Pigmentation of the skin, occurring in patches, which may be found on any part of the body. The patches vary considerably in size.

(4) There are many sensory and motor phenomena, such as pains, cramps, and paralysis, due to the presence of the nerve tumours, and, in addition, marked mental changes appear, including loss of intellectual power and difficulty in speaking.

The **Prognosis** depends on the possibility of the removal of the tumours which are causing the pain or paralysis just referred to.

3. **PLEXIFORM NEUROMATA** are tumours made up of a tortuous bunch of nerve fibres, with often a considerable amount of myxomatous tissue around the constituent nerve bundles. They are frequently met with in connection with the 5th nerve, but occur on many of the nerves of the body. They are almost invariably the result of a developmental error.

4. **AMPUTATION NEUROMATA.**—When a limb has been amputated, a bulbous swelling forms on the central end of the divided nerves. These swellings consist largely of axis-

cylinder processes which have grown downwards and become convoluted, and they are surrounded by a network of fibrous tissue. The existing axis-cylinders at the terminal end of the divided nerve unquestionably form numerous young axis-cylinders by a process of fission, so that in the neuroma there are actually more axis-cylinders than in the nerve just above the level of section. These neuromata cause pain if they are so situated as to be pressed upon in the stump, and it is prudent in performing amputations to divide the nerves as high up as possible. Occasionally a painful amputation neuroma requires to be removed.

Carcinoma and sarcoma may also occur on nerves, but with the exception of the tumours above mentioned no other neoplasm requires reference.

6. NEURALGIA

NERVE PAIN.—It may be associated with definite inflammation of the nerve affected, or there may be no recognisable pathological change present. It may, therefore, be of organic or purely functional origin. Pressure on a nerve may also cause pain.

Etiology.—The functional cases are common in debilitated and anæmic persons, and in neurotic and gouty individuals, and they frequently follow in the train of a continued fever, and especially influenza; while the immediately exciting causes include a carious tooth, cold, the act of mastication, gastric disturbances, and so forth.

Pathological Anatomy.—Although there is usually no visible change in the affected nerves in so-called functional cases, there may be swelling of the nerve and even changes in the central cells.

Clinical Features.—The pain is aching or lancinating; it may be paroxysmal and almost unbearable, or comparatively slight. There are painful points corresponding to the positions of foramina through which the nerve emerges, and the affected skin may be swollen and hyperæsthetic. The hair belonging to the area may even change colour, and there may be local sweating. The pain lasts for hours or for a whole day, and there is often a periodicity about its recurrence.

TYPES OF NEURALGIA.—(1) NEURALGIA OF THE 5TH NERVE.

TIC DOULOUREUX.—(Compare diagram of nerve distribution on page 577.) Any of the three divisions may be involved. When the ophthalmic division is affected, supra-orbital pain is the most common manifestation, and pressure over the supra-orbital notch greatly aggravates it. The conjunctiva is often inflamed, and tears may flow freely. Not infrequently herpes frontalis is associated, and even iritis. Almost in every case the eyeball is painful. Where the second or superior maxillary division is involved, pain is felt over the point of emergence of the nerve at the infra-orbital foramen, and the upper teeth on the affected side are painful. In these cases, the existence of dental caries and the condition of the antrum should be investigated. Where the third or inferior maxillary division is involved, there is often pain felt either in connection with the mental foramen and lower teeth, or the auriculo-temporal branch behind the ear. Not uncommonly there is twitching of facial muscles associated with very severe cases.

Paroxysmal cases of trigeminal neuralgia are called *epileptiform*, and the almost continuous paroxysms of agonising torture give scope for the ingenuity of the physician in trying to afford relief.

The **Diagnosis** really implies in most cases an attempt at discovering the cause of the pain.

The **Prognosis** depends on the recognition of the cause and its treatment, successful or the reverse.

The **Treatment** is twofold. Find out any definite causal factor, and treat it. Give iron and arsenic for anæmia, salicylates for rheumatism, colchicum for gout, and quinine for malaria. Examine the nose and teeth, treat any gastric disturbance present, and attend to the general health.

The special remedies for the relief of the pain are the analgesic group, of which the best are—quinine (gr. 5-10), antipyrine (gr. 10-20), phenacetin (gr. 20), antikamnia (gr. 5-15), butyl-chloral hydras (m 10-20), and exalgin (gr. 2-3). Aconite, bromides, and tincture of gelsemium are also useful. Always be careful to avoid depressing the patient too much; in certain cases a diffusible stimulant, and especially alcohol, works wonders. Locally, chloral and camphor in equal parts may be painted on, or menthol, veratrine, or aconitine ointments rubbed gently over the site of the pain. Local hypodermics of osmic acid (1 or 2 drops of

a 1 per cent solution), cocaine, or morphia are sometimes used. Galvanism is worth a trial, the positive pole being applied to the painful part, and the negative pole placed over the vertebral column.

In severe and persistent trigeminal neuralgia, nerve section, or even the removal of the Gasserian ganglion, has been recommended.

Neuralgia of Certain of the Spinal Nerves.—Many of the spinal nerves may give rise to neuralgic pain, the initial and causal lesion being tumours, injuries, pressure, syphilis, and, in some cases, exposure to cold. Certain of these neuralgias demand special reference, and many of them are in reality cases of neuritis.

(2) NEURALGIA OF THE BREAST, MASTODYNIA.—This is a neuralgia of the branches of the intercostal nerves supplying the skin or the gland structure of the breast. It may result from excessive stimulation, as in over-lactation or in pregnancy, but it is also not infrequently associated with menstrual irregularity or hysteria. There are generally tender points suggestive of the cutaneous distribution of the intercostal nerves. Common-sense treatment, with the local use of sedatives, affords relief.

(3) BRACHIAL NEURALGIA.—This form of neuralgia is sometimes the result of an injury, but is more likely to occur in persons predisposed by rheumatism, gout, anæmia, or debility. The pain may be very excessive. It should be remembered that pianists' and telegraphists' and other trade cramps are often associated with neuralgic pain, specially experienced in the nerves of the brachial plexus. The treatment should be on general lines for neuralgia; sometimes local counter-irritants or sedatives are efficacious, although in many cases rest to the affected arm is of the greatest value.

(4) INTERCOSTAL NEURALGIA is almost invariably diagnosed by the three painful points or areas which correspond to the dorsal, lateral, and anterior cutaneous branches of the nerve. Very commonly herpes zoster is associated with the pain, and especially in cases of great severity and long duration.

(5) LUMBO-SACRAL NEURALGIA.—Just as in intercostal neuralgia there are usually three painful points, so the cutaneous distribution of the nerves of the lumbar plexus often gives rise to pain in the three positions where the super-

facial branches come to the surface—that is to say, (1) close to the spinal column, (2) in the lateral region, and also (3) anteriorly, near the middle line.

The causes are various. There are generally *predisposing* causes, such as anæmia, ill-health, or neurasthenia, and also local or *exciting* causes, such as a pelvic tumour, constipation, etc.

The **Diagnosis** is by no means easy. The physician has to remember the possibility of some malignant growth, and that the neuralgia very frequently has an organic cause.

(6) SCIATICA may be either a purely functional neuralgia, or it may be a definite perineuritis or interstitial neuritis.

Etiology.—It is most common in adults and in males. It is closely associated with rheumatism and gout, and certainly exposure to cold or wet in a rheumatic subject is extremely apt to induce an attack. Many cases of sciatica are really interstitial neuritis and not merely a functional neuralgia.

Pressure on the sciatic nerve, whether in the pelvis or on the sacral nerve roots, or less probably in the line of the nerve, causes sciatica; but although the nerve may actually be compressed, the pain is generally of neuralgic nature, and not due to a definite neuritis. Constipation, pelvic tumours, and the pressure of the fetal head are common causes of such sciatic pain. It is also probable that, just as a carious tooth may set up a trifacial neuralgia, so a pelvic tumour may give rise to a reflex neuralgia of the sciatic nerve. It is doubtful whether syphilis is often an important factor.

Pathological Anatomy.—If there is neuritis, the nerve trunk is generally swollen, and the exudations already mentioned may be found interstitially either inside or outside the funiculi, while hæmorrhages are not infrequent. These changes are more commonly met with at the sciatic notch, or in the middle of the thigh. Where the pain is the result of pressure, or is a true neuralgia, there may be no recognisable anatomical change.

Clinical Features.—The chief clinical feature is pain. It comes on gradually, increasing in violence towards nightfall. It is greatly increased by muscular effort, or by pressure over the nerve. The patient cannot stoop, nor is he able to sit on a hard seat without suffering torture, and he walks with affected knee bent. The pain may be burning, boring, or

shooting, and may shoot down to the knee, possibly to the heel. There are certain specially painful points: (1) above the hip-joint, (2) in the popliteal space just above the head of the fibula, (3) behind the external malleolus, and (4) over the dorsum of the foot. Cramps not infrequently occur in the affected muscles, and the muscles may waste. There may be a certain amount of cutaneous anæsthesia. Sciatica is an obstinate and long-standing affection, very refractory to treatment, and relapses occur notwithstanding every possible care. In severe cases the patient is confined to bed altogether.

The **Diagnosis** is aided by the digital investigation of the nerve, and the chief point of importance is to decide what is the cause of the condition. The utmost care should be taken to investigate the pelvis and also the hip-joint. Where there is pressure on the cauda equina, the pain is generally bilateral.

The **Prognosis** as a rule is favourable; but protracted cases generally cause muscular atrophy, and imply a damage to the nerve which it may take many months to recover from.

Treatment.—In an acute case the limb should be kept absolutely at rest, and, if possible, a splint applied. Locally much may be done by poultices, or sometimes by blisters, to relieve the aching pain. The button cautery is often of service; but probably in an obstinate case, where it is not simply a neuralgia, but a true interstitial neuritis, acupuncture yields by far the most satisfactory results. The line of the nerve should be marked out accurately, and the needles, half a dozen in number, should be rendered aseptic, and then inserted at intervals of about an inch into the nerve; the highest needle should not be above the fold of the buttock, and the lowest must not reach as far as the popliteal space. The needles may be left in position for half an hour or thereby, the limb being covered by a cage so as to remove the pressure of the bed-clothes. The object of the treatment is undoubtedly to permit escape of some of the exudation from the nerve sheath; and when we remember that the lymphatics inside the nerve run for considerable distances without communicating externally, the value of this treatment, where an exudation is present, is obvious. Sometimes a 1 per cent solution of osmic acid (m 2-10) is injected into the nerve with good results as regards pain, although the conductivity

of the nerve is thereby greatly affected for some time. Internally rheumatism should be treated by means of salicylate of soda, and gout or syphilis, if present, should be treated on the usual lines.

Baths are often of great value, peat-baths or warm mud-baths affording much comfort to the sufferer. In certain cases high frequency has proved efficacious, but galvanism is disappointing. Massage should be used for wasted muscles, but only after the inflammatory stage has subsided.

Lastly, reference must be made to the surgical procedures of removing a portion of the sciatic and of nerve-stretching. It seems probable that the removal of a part of the nerve permits of the escape of exudation, and therefore in some cases is advantageous. Nerve-stretching has rather waned in favour of late years. The tension used to the nerve is sufficient to lift the limb off the operating table. Unquestionably by this means adhesions may be broken down, but only in a limited number of cases is the result successful.

(7) PLANTAR NEURALGIA.—This is not uncommon in flat-foot, the pain being largely due to pressure on one or other of the branches of the nerve in the sole of the foot.

(8) METATARSAL NEURALGIA, OR MORTON'S AFFECTION OF THE FOOT.—This consists in spasmodic attacks of pain starting at the base of the fourth, sometimes of the second toe, and shooting up the leg. It is increased by pressure over the head of the metatarsal bone.

The affection is due unquestionably to tight shoes, which exert pressure on the heads of the metatarsal bones, especially the fifth, and persons tending to flat-foot often suffer. The treatment is very simple—broad shoes, the use of a special spring in the boot so as to raise the instep, and, where these measures fail, excision of the head of the metatarsal bone.

(9) TESTICULAR NEURALGIA, COCCYGEAL NEURALGIA, ETC.—Neuralgic pain may also be felt in the region of the testicle or spermatic cord, in the rectum, and in the urethra, while a coccygeal neuralgia, which is often of great severity, is due to pressure on the coccyx. This coccygeal pain is apt to follow labour or a severe injury to the coccyx. Many hysterical patients suffer from pains of this kind, and the treatment consists largely in an attempt to combat the hysteria and to apply sedatives locally. In several cases we have seen

benefit from applications of the high-frequency currents to the region of the rectum.

(10) VISCERAL NEURALGIAS. — The crises in locomotor ataxia, gastric, œsophageal, etc., are neuralgias, and angina pectoris may also be neuralgic in nature. Pelvic neuralgia is not uncommon in women, especially in hysteria.

Erythromelalgia, or red neuralgia, implies pain, redness, and swelling, and affects either the hands or feet. It resembles in certain respects Raynaud's disease, and is described later.

7. DISEASES OF THE SPINAL NERVES

(1) *Paralysis of the Phrenic Nerve*

This may be the result of peripheral neuritis (diphtheritic or alcoholic), or it may be due to pressure on the nerve, as from tumour. The phrenic nerve arises from the 4th and 5th cervical segments, and these may be compressed or injured, or the anterior nerve roots springing from them may be the site of the lesion.

Clinical Features.—Paralysis of the diaphragm in most cases is bilateral, and breathing is entirely confined to the thoracic muscles. In place of distension of the abdomen on inspiration there is retraction, and, on the slightest exertion, extreme dyspnoea is experienced. Coughing is difficult, and bronchitis is a common sequel. If the paralysis is unilateral, the absence of movement on the affected side of the epigastrium is very typical.

The **Diagnosis** is generally fairly easy, although a mild degree of paralysis may present considerable difficulty.

The **Prognosis** depends on the cause, but certainly in cases of peripheral neuritis, whether of alcoholic or diphtheritic origin, a fatal result is extremely probable.

The **Treatment** may necessitate an attempt at artificial respiration, especially where, as in peripheral neuritis, the intercostal muscles also become involved. Oxygen inhalations are to be commended.

Hiccough.—Hiccough is the result of spasmodic contraction of the diaphragm. Peripheral stimuli, frequently

from the stomach or from the region of the abdomen, act reflexly on the respiratory centre, while in other cases the stimulation may be described as central in origin, as is Bright's disease or diabetes. There are also cases of hiccough which are associated with hysteria, epilepsy, and cases in which there is local cerebral irritation. Whether the hiccough is due to peripheral or central stimuli, it is by the phrenic nerves that the motor impulses are conveyed. Associated with the diaphragmatic spasm is a sudden closure of the glottis induced by stimuli conveyed through the medium of the vagus.

Transient hiccough is readily cured by sipping cold water, a number of sips being taken as rapidly as possible without drawing breath. In serious cases, in which hiccough goes on for days or weeks until the patient is exhausted, the skill of the physician may be taxed. Sometimes the application of a mustard leaf or of an ice-bag over the stomach affords relief; in other cases it is requisite to administer powerful sedatives, such as morphia or bromides. Washing out the stomach may in certain cases be found to be successful.

(2) *The Brachial Plexus*

There are many possible forms of injury which may damage one or more cords, or the whole of the brachial plexus, such as a severe blow or fall, or the fracture of ribs or clavicle, and, in a considerable number of cases, traction on the arm has resulted in the tearing through of certain of the spinal nerve roots. Such cases have been met with in factories, where an arm has been drawn into the machinery; and similar lesions have ensued from traction on the arm at birth. It is obviously the lower nerve roots belonging to the plexus which are most likely to suffer, the roots being tightly stretched over the lower edges of the ribs close to the spinal column.

(a) **ERB'S PARALYSIS**, or the Upper Arm Type of Paralysis, is a lesion of the 5th and 6th cervical roots.

Etiology.—It may be produced by injuries such as those just referred to, by pressure above the clavicle, as from carrying a heavy load on the shoulder, and by traction on the neck of the infant at birth.

The **Clinical Features** include paralysis of the following muscles:—The deltoid, biceps, brachialis anticus, supinator

longus, and sometimes the supinator brevis, and the supra- and infra-spinatus. Anæsthesia is limited to the outer side of the upper and lower arm, and depends on the sensory distribution of the circumflex and musculo-cutaneous nerves. The result of the lesion is that the arm cannot be abducted, cannot be flexed at the elbow-joint, and supination is impossible. Pain may also be present.

(b) **KLUMPKE'S PARALYSIS**, or a Form of Lower Arm Type of Paralysis, is a lesion of the 8th cervical and 1st dorsal roots.

Etiology.—It is produced by tumours, syphilitic meningitis, sometimes by primary neuritis, and also by traction on the arm in accident cases.

The **Clinical Features** include paralysis of the small muscles of the hand and the flexors of the fingers in the forearm, and should the 6th and 7th cervical roots be also involved, the extensors, triceps, and the pronators and flexors of the wrist will be affected. The results of the lesion are wasting of the muscles above mentioned, pain, hyperæsthesia, or anæsthesia in the realm of the ulnar nerve, and from the affection of communicating root branches of the 1st dorsal root there is myosis, sluggish pupil reaction, and diminution in size of the palpebral fissure, the eye appearing to be somewhat sunken.

(c) **PARALYSIS OF THE LONG THORACIC NERVE, OR SERRATUS PARALYSIS.**

Etiology.—The nerve may be injured by pressure or by a punctured wound, or sometimes it is affected in neuritis.

The serratus magnus muscle fixes the scapula when the arm is raised above the horizontal, and carries the scapula outwards, forwards, and slightly upwards. When the arm is raised vertically, the scapula being fixed by the rhomboids and other muscles, the serratus can raise the ribs and help in expanding the chest. There is often paralysis of the trapezius as well.

(d) **PARALYSIS OF THE CIRCUMFLEX NERVE.**—This nerve is derived from the posterior cord of the brachial plexus, and supplies the deltoid and the teres minor muscles. It is often injured by the pressure of a crutch, and forms one of the so-called typical crutch palsies. The arm cannot be raised, and the affected muscles waste.

(e) **PARALYSIS OF THE MUSCULO-SPIRAL NERVE.**—This nerve may also be injured by the pressure of a crutch, or by hanging

the arm over the back of a chair. Fractures and injuries may also produce paralysis.

The **Clinical Features** depend on the position of the lesion, but may include paralysis of the triceps, the supinators, and the extensors of the wrist, fingers, and thumb. As a result of the paralysis, the elbow cannot be extended against pressure, the hand is pronated, and there is wrist-drop. In lead paralysis, the paralysis is often limited to the extensors of the wrist and fingers, the supinators escaping. There is limited anæsthesia of the outer part of the forearm and the dorsal aspect of the thumb, first, middle, and half of the third fingers (excepting the ungual phalanges).

(*f*) PARALYSIS OF THE ULNAR NERVE.—It arises from the last cervical and the 1st dorsal roots.

Etiology.—Injuries in the region of the elbow, sometimes of the shoulder, cause this paralysis.

The **Clinical Features** include paralysis of the muscles supplied by the nerve. These are the flexor carpi ulnaris, the flexor profundus digitorum (one-half), the interossei, the two ulnar lumbricales, the hypothenar muscles, the adductor pollicis, and the deep head of the flexor brevis pollicis. The typical claw-like hand develops after prolonged paralysis, and the chief characteristics are the over-extension of the metacarpophalangeal joints and flexion at the inter-phalangeal joints. The ulnar side of the hand, including the little and ulnar half of the third fingers, is anæsthetic, but the sensory changes only extend to the wrist.

(*g*) PARALYSIS OF THE MEDIAN NERVE.—This nerve is derived from the lower 4 cervical roots.

Etiology.—The nerve may be injured by a lesion in the forearm, such as a fracture. Should the lesion be at the shoulder, other nerves generally suffer.

The **Clinical Features** include paralysis of the flexors of the fingers, except the ulnar half of the deep flexor, the flexors of the wrist, except the flexor carpi ulnaris, the two pronators, besides several of the thumb muscles and the radial lumbricales. The cutaneous supply includes the radial side of the palm, the front of the thumb and the first, second, and half the third fingers, and also the back of the first three fingers and the last phalanx of the thumb. The forearm cannot be properly pronated, and the wrist cannot be flexed except towards the

ulnar side, while the thumb cannot be opposed to the tips of the fingers.

(3) *The Lumbar Plexus*

The lumbar plexus arises from the first three lumbar roots and half the fourth, with a connecting branch from the 1st dorsal root. It is formed in the substance of the psoas muscle, and supplies the flexors and abductors of the thigh, the extensors of the knee, and the cremaster muscle. It supplies also the skin over the lower part of the abdomen, the region of the genital organs, the greater part of the internal and external surfaces of the thigh, and the internal surfaces of the leg and foot.

The lumbar plexus may be injured in whole or in part by disease of the spinal column, such as caries or tumour, by meningeal inflammations, by affections of the cord, and by lesions involving the roots or branches of the plexus. Neuritis is not uncommon, especially a neuritis secondary to tumours or other conditions.

(a) PARALYSIS OF THE OBTURATOR NERVE sometimes occurs from pressure during labour, occasionally from tumours or obturator hernia. It causes loss of power in the abductors of the thigh with inability to cross the legs, while outward rotation is impossible.

(b) PARALYSIS OF THE ANTERIOR CRURAL NERVE may occur as the result of tumour or abscess, dislocation of the hip-joint, and other conditions. The extensors of the knee are paralysed, and there is anæsthesia of the greater part of the thigh and the inner side of the leg as far as the great toe.

(c) PARALYSIS OF THE EXTERNAL CUTANEOUS NERVE.—This nerve is mainly sensory, and is affected by neuritis, sometimes of alcoholic origin. Pains are felt along the lines of distribution of the nerve, and there is tenderness on pressure where it passes under Poupart's ligament just internal to the anterior superior iliac spine. So severe may the pain associated with this neuralgia be, that excision of the nerve has been practised for its relief.

(4) *The Sacral Plexus*

The sacral plexus is formed by the first 3 sacral nerves and part of the 4th. It supplies the extensors and rotators of

the thigh, the flexors of the knee, and all the foot muscles, and the cutaneous area governed by these nerves includes the skin over the buttock, the back of the thigh, the outer and posterior aspects of the leg below the knee, and the greater part of the foot.

Etiology.—The sacral plexus is apt to be injured by tumours, pelvic inflammations, and injury during parturition.

(a) **PARALYSIS OF THE SCIATIC NERVE.**—The sciatic nerve is the chief branch of the sacral plexus, and it may be injured by conditions similar to those above mentioned, but it must be remembered that neuritis is a not infrequent cause of paralysis, and that injuries to the nerve from fracture of the thigh, tumours, and so forth, may also cause sciatic paralysis. The nerve supplies the extensors of the hip, the flexors of the knee, and all the muscles below the knee, but it depends on where the lesion is situated whether the branches supplying the thigh muscles may not escape. Wasting of the affected muscles results, and there may not infrequently be trophic sores produced on the foot, and sometimes herpetic eruptions along the line of the nerve.

(b) **PARALYSIS OF THE EXTERNAL POPLITEAL NERVE** is generally the result of injuries or neuritis. The nerve supplies the long and short extensors of the toes, the peronei muscles, and the tibialis anticus, while the sensory distribution includes the outer half of the front of the leg and the dorsum of the foot.

The **Clinical Features** include foot-drop with the characteristic high-steppage gait. In old standing cases talipes equinus may develop.

(c) **PARALYSIS OF THE INTERNAL POPLITEAL NERVE.**—The nerve supplies the posterior tibial muscle, the muscles of the calf, the sole of the foot, the popliteus, and the long flexors of the toes, while the sensory distribution includes the lower half of the back of the leg, and the sole of the foot and plantar aspect of the toes.

Etiology.—The nerve may be injured by a blow or wound, and neuritis is a not infrequent cause.

The **Clinical Features** include loss of extension at the ankle-joint; adduction of the foot becomes impossible, and the patient cannot rise on tiptoe, while talipes calcaneus may develop from a long-standing affection.

Many of the lesions of the plexuses and nerves above mentioned are associated with more or less severe neuralgia and these neuralgic pains may be the result of irritation by pressure or of the action of toxins, while more trivial cases may depend on deficient amount, or poor quality of, the blood-supply.

8. DISEASES OF THE CRANIAL NERVES

(1) 1st Cranial Nerve—the Olfactory Nerve

Anosmia, or loss of smell, may result from lesions of the



of the accredited olfactory centres situated, according to Ferrier, in the uncinate convolutions.

Catarrhal affections of the mucosa of the nose and polypi are common causes of anosmia, while the olfactory bulbs and their filaments may be damaged by fractures in the anterior

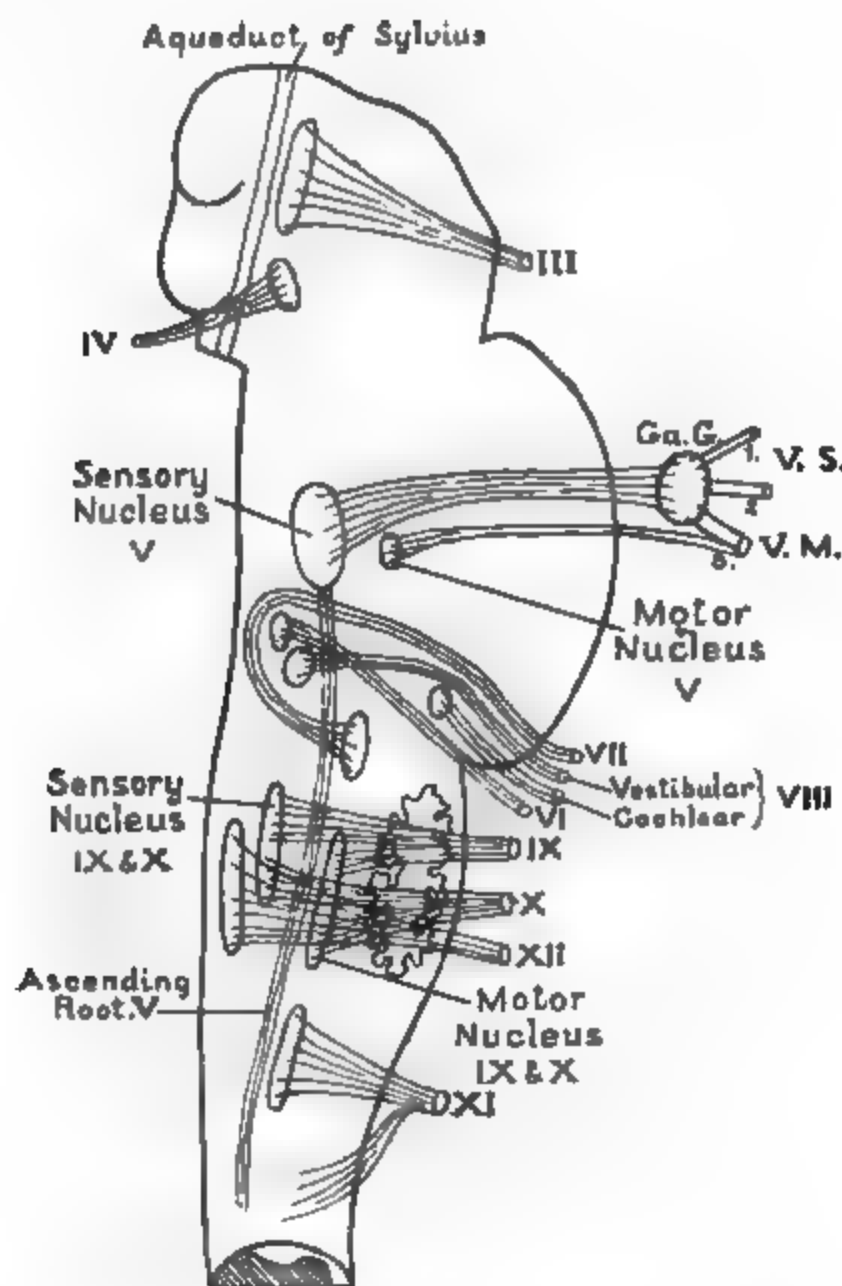


DIAGRAM 31.—Diagrammatic Representation of the Nuclei of the Cranial Nerves. The descending root of the 5th Nerve has been purposely omitted.

foosa of the skull by blows on the head, falls on the occiput (by contrecoup), caries of neighbouring bones, meningitis, tumours, and rarely by neuritis or by primary degeneration such as occurs in locomotor ataxia. Lesions of the uncinate convolution cannot be considered as a common cause of anosmia.

Smell is responsible for much of the pleasure usually referred to the sense of taste. It may be tested by sniffing oil of peppermint or other essential oil, but care must be taken

of the optic thalamus. From these ganglia the three bundles pass into the corresponding optic tract, and reach the optic commissure or chiasma. The outer half of the fibres passes to the outer half of the retina in the optic nerve of the same side, while the inner half of the fibres of each tract decussates in the commissure, and passes to the optic nerve and the inner half of the retina of the opposite side. Thus the right optic tract supplies the right halves of both retinae. The fibres supplying the macular portion of the retina, which originate in the neighbourhood of the calcarine fissure, also participate in this division at the chiasma, and pass in each optic nerve in a definite bundle—the papillo-macular bundle. The nerve fibres which pass through the external geniculate body are the most important sight fibres, while the bundle passing to the anterior corpus quadrigeminum carries upwards from the eye the impulses and forms the sensory limb of the light-reflex arc, and from the anterior corpus quadrigeminum these impulses pass by Meynert's fibres to the nucleus of the 3rd nerve, to reach the part of the nucleus functioning the sphincter pupillæ muscle. The motor limb of the arc is the 3rd nerve.

Wernicke's Pupil Reaction is the test used to determine whether this light-reflex arc is intact. In other words, it determines whether the lesion involves the optic tract, or is situated behind the anterior corpus quadrigeminum. The patient is seated in a dark room, and the ray of light must be thrown, by the ophthalmoscopic mirror, on to the blind half of the retina. If the pupil contracts, the light-reflex arc is intact; if it does not, the lesion must be situated in the optic tract. The reader is reminded that in locomotor ataxia the fibres which connect the anterior corpus quadrigeminum with the nucleus of the 3rd fibres, commonly known as Meynert's fibres, are degenerated, and so the typical Argyll Robertson phenomenon is obtained.

A lesion of one occipital lobe, of one optic radiation of Gratiolet, or of one optic tract, causes *homonymous hemianopsia*, the two corresponding halves of the retinae being involved and producing blindness of the opposite halves of the fields of vision.

A lesion through the middle of the chiasma, such as might result from pressure of an aneurismal dilatation of one of the anterior cerebral arteries, may cause the involvement of the

two nasal halves of the retinae which correspond to the two lateral halves of the fields of vision. Where the two temporal halves of the retinae are affected, a double lesion must be present, one on either side of the chiasma, and involving the two halves of the optic tracts which do not decussate. Such a double lesion causes blindness in the two nasal halves of the field of vision. Where the two nasal or the two temporal halves of the retinae are involved, and corresponding blindness results, the lesions are termed *heteronymous hemianopsia*.

If the lesion involves the angular gyrus, the resulting changes vary somewhat. There may be hemianopsia, sometimes crossed amblyopia, and, if the lesion be on the left side of the brain in a right-handed individual, there is visual aphasia or word-blindness.

It is impossible to refer here to all the forms of blindness in the visual field, but it may be desirable to remind the reader of the significance of the following terms:—

Scotoma is limitation of the field of vision. It may be peripheral or central. Peripheral scotoma is a common result of optic neuritis, whereas chronic retrobulbar neuritis often causes central scotoma. Generally the visual field for colour is seriously affected in cases where the field for white is limited; it is interesting to know that a great deal depends on the size of the test object used, and Bjerrum has, by his delicate methods of testing the vision field, greatly added to our accuracy of observation, the size of the test object used being an important feature in his method.

Amaurosis is complete blindness, temporary or permanent, without perceptible ocular lesion.

Amblyopia is loss of acuteness of vision, and is very frequently associated with toxic conditions such as tobacco poisoning.

Night-blindness is a peculiar functional condition, in which the patient fails to be able to see clearly at night.

Day-blindness is the opposite of the preceding, and is a condition in which objects cannot be seen comfortably in bright light.

(a) OPTIC NEURITIS.—Inflammation of the optic nerve, and specially the papilla, often associated with a greater or less degree of retinitis.

Etiology.—It is most frequently the result of (1) cere-

bral tumours, and occurs in 80 per cent of all such cases. Certain kinds of tumours are more apt to produce it than others, glioma taking the leading place, and the site of the tumour has a great influence. It is most common in cerebellar tumours and in neoplasms affecting the corpora quadrigemina, while it is rare in tumours of the medulla. The size of the tumour has little to do with its presence. In a big tumour optic neuritis may be absent, and in a small tumour situated in the cerebellum, and no larger than a hazelnut, it may be very intense and of rapid development.

(2) Meningitis is reputed to be the second most frequent cause, but this is doubtful. It is very rare in simple meningitis, more common in basal meningitis of tubercular origin, especially if there are definite tubercular tumours in the brain, and it is most common in syphilitic meningitis with gummata involving brain tissue.

(3) In aneurisms of the internal carotid, in abscesses and cysts, optic neuritis may occur, and it is sometimes associated with thrombosis of the cavernous sinus.

(4) It results from a group of cases to which the term toxic is applicable, and of which Bright's disease, generally the chronic form, is the best example; while in the same group we might include its rare occurrence in cases of continued fever, in lead poisoning, etc.

(5) It results sometimes from local eye disease, to which the optic neuritis is secondary, and we should especially note here hypermetropia with astigmatism.

(6) In cord and brain disease, such as myelitis, disseminated sclerosis, cerebritis, and general paralysis, it very occasionally occurs.

(7) In meningeal hæmorrhage, where the intersheath space is involved, optic neuritis may develop secondarily.

(8) It occurs sometimes as the result of anæmia, chlorosis, and other blood diseases, and it may constitute a grave danger to the patient's sight in cases of menstrual irregularity.

Pathological Anatomy and Pathology.—There is sometimes definite "choked disc," but very often the papillitis or swelling of the papilla is moderate in degree. Exudation of inflammatory lymph is always present, and there is a certain, but as a rule a limited, number of inflammatory cells. In the optic nerve the increase of nuclei is not generally a leucocytic

one, but is due, we believe, to a proliferation of neuroglial cells. There is sometimes a great cellular proliferation in the intersheath space. There may be a great increase in the fluid contents of the intersheath space, but this is by no means always the rule; when it is present, it certainly tends to produce a very marked choking of the disc, from interference with the outflow of blood and lymph from the papilla. The retina always suffers, but the retinitis varies much in extent and degree. Where recovery does not occur, optic atrophy of the consecutive type supervenes.

The backward pressure theories of optic neuritis include venous pressure in the cavernous sinus and lymph pressure in the intersheath space and the lymph spaces which communicate with it. The free communication of the cavernous sinus with orbital and facial veins, and the fact that often there is no retrobulbar distension of the intersheath space, put these theories out of court in most cases. Leber has suggested a toxic agent in the cerebro-spinal fluid, and Galezowski a toxic agent acting by continuity of tissue from the site of the tumour, abscess, etc.; others have suggested a descending neuritis, or a meningitis, commencing near the tumour and extending by the meninges to the optic nerve. We favour the toxic theory owing to various facts concerning the neuroglial hypertrophy and proliferation which cannot be discussed here.

Clinical Features.—The ophthalmoscopic appearances are very marked.

(1) At a very early stage there is simply congestion of the disc, which later forms a definite swelling, great or small in amount. The edge of the disc becomes blurred, and the sharp distinction between retina and papilla is lost, while the physiological cupping disappears, being filled up with inflammatory exudation.

(2) The vessels are more or less covered with exudation, and, as the result of pressure, the veins become enlarged and tortuous, and the arteries narrower than normal. There may be a certain amount of exudation extending along the vessels into the retina for some distance.

(3) Hemorrhages, either in the swollen disc or the retina near the disc, are not infrequent. They are generally flame-shaped, but, if in the nerve cell layers of the retina, they may be round or oval.

The visual changes vary. Sight may be preserved for a marvellously long time after optic neuritis has commenced. It may fail gradually or suddenly. There is generally peripheral contraction of the field, and more rarely central scotoma. The colour vision is specially affected, green and red suffering most markedly.

These changes, ophthalmoscopic and visual, are generally bilateral, unless they are due to an ocular cause, when they may be limited to the affected eye. When bilateral, they are often more advanced in one eye, and in tumour cases are sometimes worse on the side of the lesion. Recovery may take place even in severe cases if the cause of the condition is removed, but it is generally found that severe optic neuritis goes on to consecutive atrophy and complete loss of sight, the disc becoming white and filled in, and the vessels diminishing in size, although the veins remain proportionately larger than the arteries.

The other clinical features depend on the cause of the condition, but headache is commonly present.

Diagnosis.—It is the cause of the optic neuritis which demands investigation. There is usually headache, vertigo, and cerebral vomiting in cases of intracranial tumour, besides any localising phenomena which may be present. In Bright's disease the urine should afford a clue to the diagnosis, and in anæmia and other blood affections the examination of the blood ought to be sufficient. Hypermetropia with astigmatism is easily recognised, and menstrual irregularities afford a suggestive history.

The **Prognosis** is favourable where an intracranial tumour is operable and the optic neuritis is not too far advanced, and in syphilitic cases there is always a better prognosis if suitable medicinal treatment is commenced at a sufficiently early date.

Treatment.—In syphilitic cases give potassium iodide in 10-30 grain doses; mercury should also be tried. Operation must be attempted, where possible, for the relief of pressure, although it is definitely known that in not a few cases pressure is not the chief or only cause of the optic neuritis. In inoperable cases it is therefore considered desirable to trephine, simply because, in a proportion of patients, benefit results as regards sight. Puncture of the intersheath space has been attempted, but without much success, and lumbar puncture has yielded

disappointing results. If it can be definitely proved that the condition is toxic and the toxin is recognised, something may be possible when our knowledge is more precise, but obviously many inoperable intracranial tumours must prove fatal at an early period, and the loss of sight often only precedes death by a short period of time.

(b) CHRONIC RETROBULBAR NEURITIS.—This is the involvement by toxins of the special bundle of optic nerve fibres which supply the region of the yellow spot, and which are called the papillo-macular bundle. It is due to tobacco and alcohol, but apparently some other factor is also necessary. It is found in certain cases of cord disease, such as locomotor ataxia and disseminated sclerosis, where tobacco and alcohol have been used in great excess, and it is present as the result of other toxic agents such as diabetes, lead, syphilis, and certain drugs, such as quinine. It is a parenchymatous degeneration of the nerve fibres, and it is bilateral. There is little change in the disc at first, but later the affected part (towards the temporal side) becomes swollen and blurred, and when atrophy has developed, it is extremely white.

There is marked central scotoma—the periphery of the fields being fairly normal. This form of optic neuritis has a favourable prognosis if the alcoholic and tobacco elements in the case can be removed. Treat the condition by stopping tobacco and alcohol absolutely, and give strychnine, iron, and other tonics. Potassium iodide is often recommended.

(c) OPTIC ATROPHY.—This may be (1) *consecutive*, (2) *primary*, or (3) *secondary*.

(1) *Consecutive atrophy* is the result of optic neuritis where recovery does not occur.

The disc is filled in and white, but the edges are slightly blurred, and white lines along the vessels may indicate the site of old exudation. The veins are dilated and tortuous, although less so than in the pre-existing neuritis, while the arteries are even smaller in size than in that condition. There is complete blindness eventually, although there may be merely limited visual acuity for some time. The nasal half of the field of vision is usually first involved.

(2) *Primary atrophy* is often associated with certain cord diseases, and especially locomotor ataxia and Friedreich's ataxia, but it may develop in syphilis, diabetes, menstrual

irregularities, and in cases where cold, tobacco, alcohol, and lead poisoning are the causal factors, while it may also be congenital or hereditary. Retrobulbar neuritis, described above, might be often more correctly termed a primary atrophy limited to the papillo-macular bundle. The ophthalmoscopic picture is very different from consecutive atrophy. The disc is sharp to begin with, and of a peculiar greenish-grey colour; the lamina cribrosa is well seen in the floor of the physiological cup. The vessels, although eventually diminished in size, show no proportionate decrease in lumen. The field of vision often shows at first a central scotoma, and the colour vision is markedly interfered with at an early stage.

(3) *Secondary atrophy* is a true descending degeneration, and is due to a lesion of optic nerve, tract, or commissure, with, as a secondary result, atrophy of the fibres cut off from their central trophic cells. It may be the result of a tumour, aneurism, suppuration, fracture, or, in fact, anything exerting pressure, one occasional cause being such distension of the third ventricle, as in hydrocephalus, which may press seriously on the optic commissure. The ophthalmoscopic appearances are not very distinctive; the vessels are contracted, but the veins are proportionately larger than the arteries. The disc is sharp cut, and there are no white lines running along the vessels into the retina, but the physiological cup may be filled in. There is very rapid interference with vision, the degree and kind of scotoma being dependent on the fibres which are affected.

The **Diagnosis** of the different forms is usually not very difficult; the first and third forms cannot well be confused.

The **Prognosis** is unfavourable in most cases, but the duration of vision, however impaired, varies greatly: in primary atrophy it may be weeks, months, or even years.

The **Treatment** consists in trying to remove the cause of the condition where that is possible; in giving iron, strychnine, and other tonics, and certainly in trying the effect of anti-syphilitic remedies.

(d) **RETINITIS**. — Retinitis more properly belongs to the domain of the specialist, and only a very brief description of forms important to the physician will be mentioned here. The nerve fibres, when they leave the disc, pass into the retina, and, as stated under optic neuritis, inflammation of the optic disc must inevitably involve, to a certain extent.

the neighbouring retina. In albuminuric retinitis, however, the retina appears to suffer primarily, and only in certain cases is there a definite papillitis.

Albuminuric Retinitis.—This occurs in chronic Bright's disease, and sometimes in cirrhosis of the kidney, but is uncommon in the more acute kidney inflammations. It is believed to be due to vascular degeneration or arteriosclerosis, as the result of which fatty changes occur in the nerve fibres near the yellow spot from deprivation of blood. These fatty-looking fibres have a peculiar radial arrangement round about the yellow spot. Associated with this condition there is generally a degree of papillitis, often with many hæmorrhages, both in the inflamed disc and in the retina.

Leucocythæmic Retinitis.—There are numerous retinal hæmorrhages, and the remaining part of the retina is of a palish colour.

Syphilitic retinitis and *retinitis pigmentosa* cannot be described here, and in fact the latter condition is of too rare occurrence to demand reference.

(3) *The Oculo-Motor Nerves, the 3rd, 4th, and 6th*

(a) PARALYSIS OF THE 3RD NERVE.—*Nucleus.*—It contains five distinct cell groups, situated in front of the aqueduct of Sylvius, and in front of the anterior corpus quadrigeminum; its posterior extremity almost reaches the nucleus of the fourth.

Point of Exit from Brain.—The two nerves come out together between the crura cerebri.

It supplies (1) Superior division—Superior rectus; levator palpebræ superioris. (2) Inferior division—Internal rectus; inferior rectus; inferior oblique.

The 3rd nerve also supplies, through the ciliary ganglion, the ciliary and the sphincter pupillæ muscles.

The nucleus for convergence and certain of the other nuclei send fibres to both 3rd nerves.

Etiology.—The nucleus may be involved by tumours, hæmorrhages, or inflammations, such as acute polio-encephalitis or degeneration, but is rarely affected without other nuclei suffering. The nerve may be involved in meningitis, gummata, and aneurism; in diphtheria and locomotor ataxia a neuritis of the nerve may be present.

The *Results of Paralysis* depend much on whether the whole nerve, one division, or merely certain muscles are affected. If complete, there is slight protrusion of the eyeball, diplopia, external strabismus, ptosis, dilatation of the pupil, and paralysis of accommodation, while the eyeball can only be moved by the superior oblique and external rectus downwards, outwards, and slightly rotated inwards.

For the effects of a lesion of the separate external ocular muscles, see table on p. 575.

Ptosis may be the result of:—(1) a congenital paralysis of the levator palpebræ muscle, in which case the condition is permanent; (2) a more or less complete lesion of the third nerve; (3) idiopathic muscular atrophy; (4) a lesion of the first dorsal root or the sympathetic fibres associated with it, which causes a pseudo-ptosis as the result of sinking in of the eyeball; (5) hysteria; (6) debility in weakly persons, especially females, in whom the condition is usually only marked on awakening from sleep.

IRIDOPLEGIA, OR PARALYSIS OF THE IRIS.—(A) *The Argyll Robertson Phenomenon*, or loss of the light-reflex, indicates a lesion of the reflex arc formed by fibres in the optic nerve, the optic tract passing to the anterior corpus quadrigeminum, and from there by fibres passing to the sphincter pupillæ centre in the nucleus of the 3rd nerve; from the nucleus the motor impulses travel by the 3rd nerve through the ciliary ganglion to the sphincter pupillæ muscle. Light must be very carefully thrown on to the retina of each eye separately. The light-reflex is lost in locomotor ataxia, general paralysis of the insane, and other diseases, and its absence may help also in localising a lesion of the optic nerve fibres *behind* the commissure. See Wernicke's pupil reaction, p. 565.

(B) *Accommodation Iridoplegia* is loss of contraction of the pupil on looking at a near object.

(C) *Cutaneous Iridoplegia* is loss of the typical dilatation of the pupil on pinching the skin of the forehead.

Cycloplegia is loss of the power of accommodation, due to paralysis of the ciliary muscle; as a result the patient cannot read or see near objects clearly.

PARALYSIS OF THE 4TH NERVE.—The nucleus lies in front of the aqueduct of Sylvius, and is just behind the 3rd nucleus. The nerve fibres, after leaving the nucleus, decussate in the

valve of Vieussens, and winding round the outer surface of the crus, pass through the sphenoidal fissure to the superior oblique muscle.

Etiology.—Tumours, softening, etc., may involve the nucleus, while the nerve may be damaged by conditions similar to those from which the 3rd or 6th nerve may suffer.

Result of Paralysis.—There is defective downward and inward movement and double vision when the patient looks downwards and outwards.

PARALYSIS OF THE 6TH NERVE.—*Nucleus.*—Situated in the floor of the fourth ventricle above the striæ acusticæ.

Point of Emergence of Nerve.—Between medulla and pons, the fibres passing through the motor pyramids.

It supplies the external rectus muscle.

Etiology.—The nerve, from its long course, is commonly affected by tumours, tubercular meningitis, gummata, and so forth.

The nuclear origin of the opposite *internal* rectus is connected with the 6th nucleus by a strand of fibres, and therefore in a nuclear lesion the opposite internal rectus is affected along with the external rectus of the side of the lesion. Further, because the fibres of the 7th nerve pass closely round the 6th nucleus, there is often facial paralysis on the same side as the lesion.

If the nerve is paralysed, there is strabismus, diplopia, and often considerable giddiness.

When examining paralysis of any of the muscles supplied by the 3rd, 4th, or 6th nerves, we should note the *primary deviation* of the affected eye and the *secondary deviation* of the normal eye, the latter being due to the effort of the patient to fix the object with the affected eye. These deviations, when measured and compared, yield important information. The secondary deviation greatly exceeds the primary in cases of paralysis, while in strabismus resulting from an error of refraction or from spasm it is usually exactly the same as the primary.

Diplopia implies the existence of a false image. When the false image is on the same side as the eye with which it is seen, it is simple or homonymous. When on the opposite side, it is crossed. The false image is always projected in the direction in which the paralysed muscle ought normally to

move the eye, and the false image is farthest from the true when the attempt is made to use the affected muscle. The patient, by turning the head, tries to avoid the diplopia, and prevent the giddiness and sickness to which it often gives rise.

The following scheme explains itself: Remember that the obliques, acting round pulleys, pull the eyeball exactly in the opposite direction to what the terms superior and inferior would suggest. Both obliques move the eyeball *outwards*, and the superior and inferior recti, counteracting this, move them *inwards*; and, while the two superior muscles (oblique and rectus) rotate the eyeball *inwards*, the two inferior muscles rotate the eyeball *outwards*. The right eye is taken as the affected one, and the arrow represents the false image.

Normal Movements.







| Muscles. | Outwards, Inwards. | Upwards, Downwards. | Rotation, inwards or outwards. | Images. (Nasal, Temporal.) |
|------------------|-----------------------|------------------------|---|---|
| Superior oblique | Outwards. | Downwards. | Inwards. |  |
| Inferior oblique | Outwards. | Upwards. | Outwards. |  |
| Superior rectus | Inwards. | Upwards. | Inwards. |  |
| Inferior rectus | Inwards. | Downwards. | Outwards. |  |
| External rectus | Outwards. | | |  |
| Internal rectus | Inwards. | | |  |

DIAGRAM 33.

To get the rotation, place a pencil first in the proper position as regards outwards or inwards, and upwards or downwards, and then, if the rotation has to be inwards, turn the point of the pencil towards the nose, if outwards, away from it.

To find the direction of the eyeball when any muscle or muscles are paralysed, delete the affected muscles; then, by scoring out the movements of the remaining muscles which counteract each other, it is easy to obtain the final result.

External Ophthalmoplegia is the term applied to more or less complete paralysis of the external ocular muscles. It may be due to a nuclear lesion or meningitis, but it is specially common in locomotor ataxia and general paralysis. It is often associated with *Internal Ophthalmoplegia*, which is paralysis of the iris and the ciliary muscle.

In conclusion, there may be a supranuclear involvement of the 3rd, 4th, or 6th nerves, which may, in rare cases, give rise to crossed paralysis.

Treatment of Ocular Paralyzes.—Try to find out the cause, and if possible treat it. Antisymphilitic remedies should be administered, and an attempt made to arrest neuritis, if present, by counter-irritation behind the ear or over the temple. Electricity is of little use: strychnine may be ordered in certain cases, and by prisms, or by covering the affected eye, much may be done to counteract the diplopia.

(b) SPASMS OF OCULAR MUSCLES.—These may be tonic or clonic.

Conjugate deviation is common in apoplexy, the patient looking upward towards his lesion in “destructive” cases, while in “irritative” cases he looks away from it. Spasms of ocular muscles may also occur in meningitis, or where there is basal irritation. In chorea there may be diplopia, and in hysteria and epilepsy there are often spasms, tonic and clonic, of ocular muscles. Serious errors of refraction may also cause spasm.

Nystagmus is clonic spasm, usually lateral, rarely vertical or rotatory. It may be intermittent and only appear when a weak muscle is put on the stretch. It varies from 60 per minute to an uncountable rate, and the magnitude of the movements also varies between coarse and excessively fine oscillations. Its causes are: (1) purely ocular, as in high

grades of refractive error, choroiditis, opacities in the lens, in albinism, and also in other congenital defects in the retina and choroid, (2) in certain lesions of the pons, cerebellum, or optic thalamus¹; (3) in insular sclerosis and hereditary ataxia; (4) in certain trades, as miner's nystagmus, in which the eyes are strained in some given direction.

The **Treatment** of nystagmus is sometimes satisfactory; where it is the result of trade-spasm, rest often effects a complete cure.

(4) *The 5th Nerve or the Trigeminal Nerve*

The course and distribution of the fibres of the 5th nerve, both sensory and motor, are shown in Diagram 34.

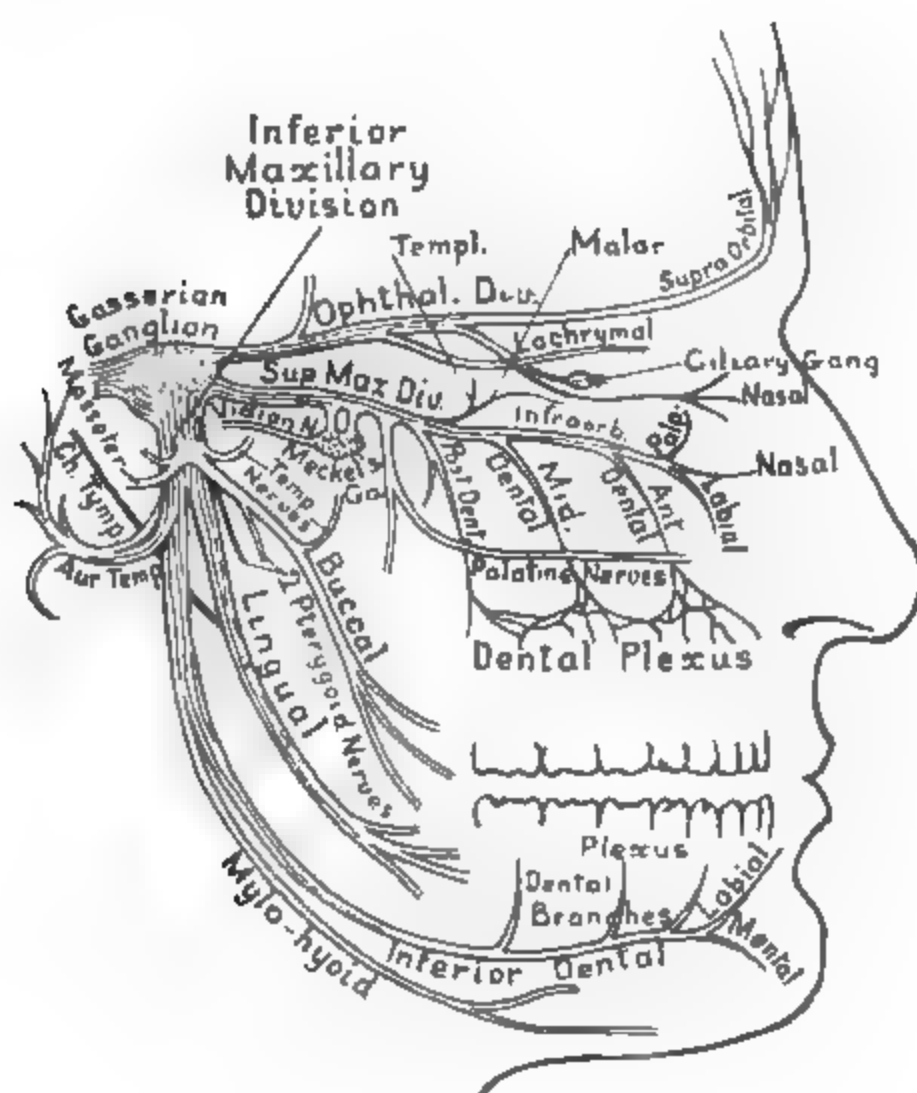


DIAGRAM 34.—Schematic Representation of the Distribution of the 5th Nerve.

(a) **PARALYSIS.**—**Etiology.**—A lesion of the nerve may be

¹ The nucleus of Deiters has been demonstrated, by the researches of Dr. Alexander Bruce, to be intimately connected with the 5th nucleus. A lesion of Deiters' nucleus or of its connecting fibres is often responsible for nystagmus.

due to the following conditions:—*Outside the skull* a secondary neuritis is not uncommon; and may be the result of a tumour, or of irritation in the region of the parotid gland or the sphenomaxillary fossa. A primary neuritis associated with cold, gout, syphilis, or other affection is rare.

At the base of the brain, tumours, meningitis, syphilis, and caries of bone may cause damage to the nerve.

In the pons, tumours, hæmorrhage, softening, and sclerosis are not uncommon; and chronic diseases, such as bulbar paralysis, tabes, and syringomyelia, may be associated with an involvement of the nerve.

The *first* division may be affected by pituitary tumours, aneurisms of the internal carotid and cellulitis in the orbit, while the *second* and *third* divisions more frequently suffer from a lesion in the sphenomaxillary fossa.

The sensory nuclei are widely separated, and part may escape where the lesion is nuclear. Supranuclear lesions also rarely involve the whole of the nerve, the motor fibres generally escaping. Paralysis of the motor fibres of the 5th nerve is often the result of a lesion of the nerve, and not of a lesion of the nucleus, while occasionally a cortical lesion results in a more or less complete involvement of the masticatory muscles.

Clinical Features.—The nerve supplies common sensation to one-half of the face, a great part of the side of the head, the conjunctiva, the mucous surfaces of half of the mouth and tongue, and one nostril. A lesion, therefore, of the whole of the nerve will cause a hemianæsthesia of the region mentioned. It is, however, rare to find such an extensive lesion. Pain is extremely common; it may be sharp and shooting, and the painful points which indicate the foramina where branches of the nerve become superficial, are typical of neuralgia of this nerve. When the patient drinks, he only feels the cup with half of the lips, this giving him the impression of a bit being broken out of the cup. The nasal mucosa is affected because it becomes dry, and, therefore, the olfactory nerve-endings are incapable of receiving proper impressions. The secretion of saliva and of tears is lessened, and where there is any irritation of the nerve, ulceration is extremely common, and may cause inflammation of the cornea with eventual panophthalmitis, while the teeth may loosen and the gums ulcerate. At first it was thought that these trophic changes

depended on interference with the Gasserian ganglion, but the ganglion has been removed for severe cases of trifacial neuralgia without any trophic changes occurring. On the other hand, herpes frequently involves the branches of this nerve, especially the supra-orbital branch of the first division, and undoubtedly it is due to trophic irritation.

Taste is supplied to the anterior two-thirds of the tongue by the chorda tympani nerve, which joins the lingual branch of the third division of the 5th, the taste fibres probably passing by the chorda tympani along the 7th nerve to the geniculate ganglion, and thence by the great superficial petrosal nerve to Meckel's ganglion, and reaching the brain by the second

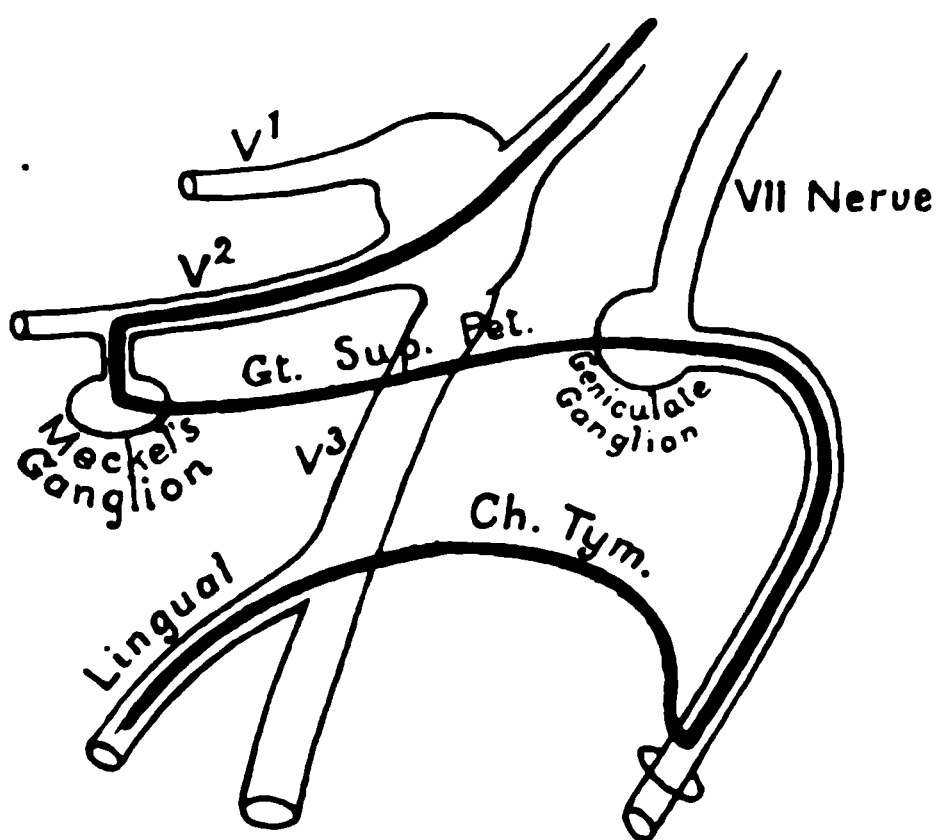


DIAGRAM 35.—Diagrammatic Representation of the course of the Taste Fibres to the anterior two-thirds of the Tongue.

division of the 5th. The taste fibres for the posterior third of the tongue, and the neighbouring portion of the palate and tonsil, are believed to pass to the brain by means of either the same division of the 5th nerve or the third division.

The motor portion of the 5th nerve supplies the muscles of mastication, namely, the masseter, temporal, and two pterygoids. The motor root passes behind the Gasserian ganglion, and its position may be seen in the diagram of the nerves at the base of the brain, while by reference to the sketch of the nuclei of the cranial nerves (Diagram 31), the relationship of the motor nucleus to the sensory will be made clear.

In paralysis of the whole motor root the patient cannot bite on the affected side, and the muscles are felt to be soft

and flabby when he attempts to clench the jaws. Food tends to accumulate between the cheek and the gums on the affected side, and the three following alterations as regards movement and position may be noted:—

(1) The lower jaw cannot be moved toward the sound side.

(2) When the lower jaw is depressed, it is displaced towards the paralysed side, and

(3) When the mouth is wide open, the condyle of the lower jaw projects markedly on the paralysed side.

The **Diagnosis** of a lesion of the 5th nerve is easy, but it may be difficult to ascertain its exact position. If the Gasserian ganglion is the site of affection, all the divisions and often the motor root are involved. If the lesion is in the pons, it is usually bilateral, and if in the posterior limb of the internal capsule, there is generally hemianæsthesia.

The **Prognosis** will depend on the nature of the lesion.

Treatment.—Pain may have to be relieved by the use of morphia or local anæsthetic preparations. Syphilis should be appropriately treated, and sometimes strychnine, together with galvanism or faradism, proves beneficial in paralysis.

Trifacial neuralgia is discussed on p. 550.

(b) SPASM OF THE MUSCLES SUPPLIED BY THE 5TH NERVE.—MASTICATORY SPASM.—TRISMUS.—Masticatory spasm may be *tonic* or *clonic*. The tonic form is exemplified in tetanus or lockjaw and the tonic spasm of an epileptic fit. Clonic spasms occur in rigors, in hysteria, and in the clonic stage of epilepsy.

(5) Affections of Taste

Loss of taste is described as *Ageusia*; perversion of the sense of taste is called *Parageusia*, and this latter condition is not infrequently due to hysteria or insanity.

Taste is closely associated with the sense of smell, and the appreciation of a good dinner and of the bouquet of wine is more dependent on the integrity of the olfactory nerve than of the gustatory.

In testing taste we must remember that by the gustatory nerve we can only distinguish (1) bitterness, (2) sweetness, (3) saltiness, and (4) acidity, and it is easy by using quinine, sugar, common salt, and vinegar, to determine whether these four primary properties of the sense of taste can be appreci-

ated by the patient. We must be careful to prevent the tongue from being drawn into the mouth after a little of one or other test solution has been applied, and the patient should indicate the taste he appreciates by writing instead of by speech. The well-known metallic taste produced by a feeble galvanic current passed through the tongue is a satisfactory test of the gustatory sense.

In lesions of the lingual branch of the third division of the 5th nerve, and of the glosso-pharyngeal nerve, taste may be abolished on the affected side; but it is still doubtful whether the taste fibres really reach the brain by the divisions of the 5th nerve, although it seems probable that the roots of the 5th nerve do carry most, if not all, of the gustatory fibres. It is remarkable in how many cases the Gasserian ganglion has been removed without producing loss of taste in any part of the tongue, and it has been suggested that the chorda tympani taste fibres may pass direct from the geniculate ganglion to the brain by the nervus intermedius of Wrisberg.

(6) *The 7th Nerve*

The motor area from which the facial fibres originate is situated in the lower part of the ascending frontal, and also in the posterior portion of the inferior frontal convolutions. From this centre, the fibres pass downwards through the internal capsule, decussate, with the exception of a few fibres, in the middle of the pons, and terminate in the 7th nucleus, which is situated in the lower part of the pons, and just internal to the ascending root of the 5th nerve. From the nucleus the 7th nerve passes round the 6th nucleus, forming a loop, and emerges just external to the 6th nerve at the lower part of the pons. The nerve, in close relationship with the 8th, enters the internal auditory meatus, and passes through the petrous part of the temporal bone, emerging from the skull by the stylo-mastoid foramen. In the petrous part of the temporal bone the geniculate ganglion is situated on the nerve, and at that point the chorda tympani fibres enter the nerve to leave it prior to its emergence at the stylo-mastoid foramen.

(a) PARALYSIS.—Paralysis of the muscles supplied by the 7th nerve may therefore be due to a lesion in several different

positions:—(1) The lesion may be cortical. (2) It may be in the internal capsule or in the pons, at any rate between the cortical centre and the nucleus. (3) The lesion may be in the nucleus of the nerve. (4) The lesion may be in the nerve itself—Bell's paralysis—and it may involve the nerve—(a) behind the geniculate ganglion, (b) between the geniculate ganglion and the departure of the chorda tympani fibres, or (c) after these fibres have left the nerve.

In supranuclear lesions the orbicularis palpebrarum and frontalis muscles are generally unaffected, and emotional movements are well seen, while in nuclear and nerve lesions all such movements are lost. This escape of the upper face muscles may be due to their innervation in part by the fibres which do not decussate in the pons.

Etiology.—The *cortical centre* may be injured by tumours, abscesses, softening, or hæmorrhage, and in the pons similar lesions may occur. In the region of the internal capsule hæmorrhage is most commonly situated in, or near, the lenticular nucleus, and as the face fibres pass down in the genu, it not infrequently happens that pressure on the internal capsule involves these fibres as well as those of the arm and leg. Thrombosis and embolism more frequently affect the cortical centre than either the tracts or the nucleus.

The *nucleus* may suffer along with other nuclei in cases of bulbar paralysis, and among the causal lesions may be either encephalitis, softening, hæmorrhage, or tumour.

The *nerve* may be affected in the skull by tumours, meningitis, or hæmorrhage, and in the petrous part of the temporal bone, middle-ear disease or fracture of the skull in the region of the mastoid sometimes involves it. Operations on the lower jaw, parotid tumours, and tubercular and other glandular enlargement in the neck, may also interfere with the nerve. Neuritis is, however, the most common cause of Bell's paralysis. It is generally interstitial, and, if so, is usually of rheumatic or syphilitic origin. Exposure to cold, and especially a chilly wind blowing on the side of the head, is a common antecedent history. Parenchymatous neuritis is most frequently of diphtheritic origin. The probable site of the lesion in cases due to exposure to cold or to syphilis is at, or near, the stylo-mastoid foramen.

A bilateral lesion implies—(1) middle-ear disease on both

sides, (2) a basal lesion, probably meningitis, and in most cases also the involvement of other nerves, (3) a bilateral lesion in the pons, and (4) a bilateral cortical lesion, which last is extremely rare.

Pathological Anatomy.—In cases of encephalitis the nuclear cells may show marked changes, similar to what is found in the motor cells of the anterior horn of the cord. No special description requires to be given of interstitial neuritis, and whether the lesion be parenchymatous or interstitial, the changes noted are similar to those described under these forms of neuritis. Where the nucleus or nerve is seriously involved, the affected muscles show marked atrophic changes; but, on the other hand, where the lesion is supra-nuclear, any wasting of muscles is the result of long-standing disuse.

Clinical Features.—In a case of Bell's paralysis, and in cases where the lesion involves the nucleus of the nerve, there is complete loss of power in the muscles affected, with, as already noted, loss of the movements representing emotional expression. The skin is smooth and the wrinkles disappear on the affected side. The eye on the affected side cannot be closed, because the levator palpebrae muscle acts unopposed, and when the attempt is made, the eyeball is rotated upwards under the lid. In time, the lower eyelid droops, and tears are apt to run down the face, because the canaliculus is not kept in accurate apposition to the eyeball. The patient cannot whistle, show the teeth, wrinkle up the face, or move the ear, and he smiles with one side of the face only. Drinking is difficult, because of the impossibility of keeping the lip closely applied to the glass, and, on chewing, the food tends to collect between the cheek and the teeth. Speech is sometimes slightly indistinct owing to the difficulty of using the lips. The palate and uvula are not affected, because the levator palati and azygos uvulae muscles are innervated by another nerve.

Taste may, or may not, be abolished in the anterior two-thirds of the one side of the tongue, this depending on whether the nerve is affected after the taste fibres have joined the 7th nerve and before they have left it in the chorda tympani nerve, or not. The involvement of taste would therefore mean that the lesion is situated in the internal auditory canal, and would most probably be due to ear disease. The stapedius

muscle is supplied by a branch from the 7th nerve, and where this branch is paralysed, the tensor tympani acts unopposed, rendering the drum of the ear unduly sensitive to loud sounds.

Where the lesion involves the nucleus or nerve, the facial muscles supplied by the nerve waste and show after a period of seven to ten days a more or less pronounced reaction of degeneration, while faradic stimulation of both nerve and muscles should be lost. In less severe cases a modified reaction of degeneration is obtained, and recovery may be hoped for in one to two months (see p. 538).

There are no sensory fibres belonging to the 7th nerve, and therefore pain is improbable, and only in a limited number of cases does a herpetic eruption appear along the line of the affected fibres.

The development of the paralysis is usually rapid when it follows exposure to a cold wind, and in most cases recovery results after a considerable number of weeks. Where only a modified recovery occurs, there may be a marked degree of contracture of the affected muscles, causing deep wrinkles on the paralysed side, and giving the suggestion, on a cursory examination of the patient, that the healthy side, being the more smooth, is the paralysed one. This contracture is sometimes extremely painful.

Where the lesion is supranuclear, the paralysis is incomplete, and usually involves the muscles of the lower part of the face. The patient can close the eye, although not quite perfectly; and the closed lid, if attempted to be raised, is found to be less resistant than that on the healthy side. Emotional movements are retained, and electrical changes are absent. If the lesion is cortical, there may be Jacksonian epileptic seizures, and if the pons is affected, crossed paralysis is sometimes present (page 646).

Diagnosis.—There is little difficulty in the recognition of facial paralysis, and it is generally easy to diagnose the position of the lesion where it is cortical, near the internal capsule, or in the nerve, once it has entered the internal auditory meatus; but it is sometimes difficult to be certain that the lesion is nuclear. It is true, however, that if one nucleus is affected, other neighbouring nuclei will likely suffer.

The **Prognosis** depends largely on the results of electrical

examination. A modified or incomplete reaction of degeneration implies a speedy, and probably complete, recovery, whereas a complete reaction of degeneration often indicates permanent paralysis.

Treatment.—In all cases where an interstitial or perineuritis is present, apply counter-irritation—either blisters or the button cauter—near the stylo-mastoid foramen. In syphilitic cases iodide of potash should invariably be given, and once the inflammatory changes in the nerve have subsided, an attempt should be made to keep up the nutrition of the affected muscles. This may be done by applying the positive pole behind the ear, and the negative over the paralysed muscles, while massage may sometimes be used. A careful examination of the ear is also necessary. The best operative interference in cases in which improvement has ceased, is unquestionably to join the 7th nerve to the spinal part of the spinal accessory. Wonderfully hopeful results have followed this treatment, although movements of the arm may produce twitching of the face, owing to the stimuli overflowing into the 7th nerve as well as passing along the spinal accessory.

(b) FACIAL SPASM.—This spasm is in part considered under the affection *tic convulsif*, and in part under such diseases as tetanus, tetany, epilepsy, and eclampsia. The movements may be twitching, clonic movements, or muscular spasms of a tonic nature.¹ They are often due to an irritant such as a carious tooth, or to exposure to cold, or may be the result of some emotional disturbance. It should, however, be remembered that there may be in the nerve itself some cause of irritation, such as a tumour or other irritative lesion.

(7) *The 8th Nerve, or Auditory Nerve*

The 8th nerve consists of two separate divisions, the cochlear and the vestibular (see Diagram 31), the cochlear specially subserving the function of hearing, and the vestibular, equilibration. The cochlear root passes to the outer side of the restiform body, and eventually becomes continuous with the striæ medullares; while the fibres of the vestibular root reach the nucleus in the floor of the fourth ventricle, which nucleus is connected with the middle lobe of the cerebellum,

¹ Twitching movements of the muscles round the eye is called *blepharospasm*.

and also with the direct cerebellar tract in the cord. The cochlear nucleus is connected with the posterior corpus quadrigeminum and the internal geniculate body, while the fibres eventually reach the cortical auditory centre in the superior temporo-sphenoidal convolution.

(a) PARALYSIS.—A lesion of the *cortical centre* on the left side causes word-deafness, and the reader is referred to the description of auditory aphasia on page 648. In addition to tumours, softening, or other affection of the cortex, the lesion may involve fibres from the cortex passing to the posterior corpus quadrigeminum, or the internal geniculate body, or the fibres from them to the cochlear nucleus.

A lesion of the *cochlear nucleus* is sometimes associated with bulbar paralysis, hæmorrhages, or softening in the medulla.

A lesion of the *auditory nerve at the base of the brain* may be due to tumours, meningitis, hæmorrhage, or fracture of the skull. In locomotor ataxia a primary degeneration of the nerve has been described, but primary neuritis does not commonly affect it.

Lesions of the *cochlear and vestibular nerves in the ear*.—Many alterations of hearing are dependent on a fault in the passages and not upon any nerve defect. A lesion of the tympanic membrane may produce either hyperacusis or auditory hyperæsthesia, or may cause a greater or less degree of deafness; associated with the tympanic membrane the ossicles must be considered, the free movement of which is essential for perfect hearing. Where there is imperfect aeration of the middle ear, deafness is not uncommon on account of the drum being indrawn, and with thickenings, or perforation of the tympanic membrane, a degree of deafness is inevitable. Tinnitus aurium is hyperæsthesia, and is mainly due to the cochlear nerve, whereas Ménière's disease is sometimes associated with an involvement of the domain of the vestibular nerve, namely, the semicircular canals and sometimes the labyrinth supplied by the cochlear nerve. In the latter case tinnitus is a prominent feature of the attacks.

(b) TINNITUS AURIUM.—Hissing, whistling, chirruping, humming, or thumping sounds heard in one or both ears. They frequently increase when the patient is in noisy surroundings. The thumping or pulsating type of sounds is synchronous with the pulse, and this is very commonly the result of atheroma in the internal carotid. It should be remembered that small

or large veins may cause rushing sounds in anæmia, and it is probable that the lateral sinus is largely responsible for these sounds, but changes in the labyrinthine fluid may permit of the labyrinthine circulation becoming audible to the patient, and the cinchonism due to quinine and salicylates is of this nature. Amongst the common causes of tinnitus aurium is the presence of wax pressing on the drum, thickening of the drum, and ankylosis of the ossicles of the ear. Sometimes choreic twitching of the stapedius muscle becomes audible to the patient, and, in other cases, the gurgling sounds of fluid may be heard where an effusion is present in the middle ear. Intracranial tumours may interfere with the circulation of the inner ear, and tumours of the cerebellum and the pons are specially associated with tinnitus aurium. It will be readily appreciated that in many conditions, such as thickening of the drum and ankylosis of the ossicles, deafness is frequently associated with the tinnitus.

There are, however, other cases in which a mental element is present, for example, where, in an insane individual, imaginary melodies or croaking human voices are in reality hallucinations of hearing.

(c) **MÉNIÈRE'S DISEASE.**—Sometimes with severe tinnitus aurium there is vertigo and a tendency to sickness and vomiting.

Etiology.—It is in many cases the result of a hæmorrhage into the semicircular canals or other lesion of the inner ear, and it may be due to a severe blow on the side of the head, while it sometimes occurs in persons exposed to excessively loud sounds, which may damage the inner ear. Hammermen, especially those working in the inside of boilers, and artillerists, are more frequently affected than other persons.

Clinical Features.—The attacks are paroxysmal; they occur at irregular intervals, perhaps several in one day, or an attack on successive days, or in other cases an isolated paroxysm. The patient has to sit or lie down in order to prevent himself from falling. There is generally tinnitus aurium, always vertigo, and a tendency to nausea and vomiting, while in most cases a certain degree of deafness results from the hæmorrhage or other lesion. The attacks may pass off after rest, and sometimes after the total loss of hearing they gradually cease. Unfortunately this is not always the

Paralysis of the nerve may be the result of a lesion in the medulla such as we find in bulbar paralysis. Sometimes the nerve is affected by tumours or by meningitis at the base of the brain.

Clinical Features.—There is anæsthesia of the upper part of the pharynx, and certainly difficulty in swallowing food, while the loss of taste is confined to the posterior third of the tongue and the neighbouring parts of the pillars of the fauces. The course of these taste fibres has been the subject of much discussion.

(9) *The 10th Nerve, Pneumogastric or Vagus Nerve*

This nerve supplies not merely an extensive area of the alimentary tract, but also the lungs and heart, and its involvement causes wide-spread changes throughout the body.

PARALYSIS OF THE VAGUS.—Etiology.—A nuclear involvement is not uncommon in cases of bulbar paralysis, and is generally associated with a similar lesion of the nuclei of the glosso-pharyngeal, the spinal accessory, and the hypoglossal nerves. Meningitis may involve the nerve close to the brain, and tumours, aneurisms, and wounds in the neck may all cause paralysis of the nerve. Sometimes in the toxic forms of neuritis the vagus is affected shortly before death, and this may be the result of alcohol, diphtheria, and more rarely other causes. Neuromata sometimes occur on the nerve, and may interfere with its functions.

Clinical Features.—It is impossible to consider all the branches of the vagus together, and therefore we describe the results of paralysis of the different branches separately.

1. *Branches to the Pharynx.*—The vagus supplies, along with the glosso-pharyngeal, the muscles of deglutition. There is therefore, in paralysis, difficulty in swallowing, and as a result the food tends to find its way into the larynx, where it excites spasm, or it may regurgitate upwards by the nose, especially if the soft palate is involved.

Spasm of pharyngeal muscles occurs in various diseases, as, for example, in hydrophobia.

2. *Branches to the Larynx.*—The superior laryngeal branch supplies sensation to the laryngeal mucosa, and is also the motor nerve for the crico-thyroid muscle. The recurrent laryngeal nerve supplies all the other muscles of the larynx,

and these laryngeal fibres reach the brain by the spinal accessory root. The recurrent laryngeal nerve on the left side is frequently affected in cases of aneurism of the arch of the aorta, and on the right side it may be involved in pleuritic adhesions. In cases of laryngeal catarrh certain muscles tend to be paralysed, and the paralysis does not endanger life; but, on the other hand, toxic paralysis, as in diphtheria, involves the abductors of the glottis, and readily causes death by suffocation. Hysterical paralysis almost invariably affects muscles which do not endanger life.

(a) *Adductor Paralysis*.—This results from the involvement of the crico-arytenoideus lateralis muscle, or the arytenoideus muscle, or both, and it is commonly due to catarrh or hysteria. The laryngoscopic picture on attempted phonation shows total inability to oppose the cords together. Aphonia results.

(b) *Abductor Paralysis*.—This is paralysis of the crico-arytenoideus posticus, and it is commonly the result of the toxin of diphtheria. It may occur in bulbar paralysis, but is very rarely caused by hysteria or an ordinary laryngeal catarrh. The laryngoscopic picture shows total inability to open the glottis during inspiration, although there is no aphonia. This condition endangers life, as air cannot enter the chest, and there is invariably great stridor with inspiration.

Abductor paralysis may occur on one side only, in which case there is less tendency to suffocation, and, at least for a time, little hoarseness.

(c) *Paralysis of one Recurrent Laryngeal Nerve*.—This is the common result of aneurism involving the nerve on the left side. The abductor muscle is first paralysed, and the cord remains near the middle line, but later the adductors are also paralysed, and the cord becomes fixed midway between abduction and adduction in what is termed the “cadaveric” position. The sound cord crosses the middle line during attempted phonation, but the voice, as a rule, is hoarse, although there is not necessarily much difficulty of breathing.

Spasm of the muscles of the larynx is present in laryngismus stridulus, which is described separately on page 401, and also in locomotor ataxia, in which laryngeal crises are not uncommon.

3. *Branches to the Heart*.—The cardiac plexus is partly supplied by the vagus and partly by the sympathetic. The vagus retards the accelerator action of the sympathetic, and

therefore in paralysis of the vagus the heart acts with abnormal rapidity, irritation of the nerve causes greater inhibition, and therefore slowing of the heart's action. It should be remembered that paralysis of one vagus is not necessarily sufficient to cause great acceleration of the heart, but when the vagi nerves are affected by the toxins of diphtheria, influenza, and other infective fevers, very rapid cardiac action may result.

The sensory fibres of the vagus which go to the cardiac plexus may possibly give rise to the pain felt in certain cases of angina pectoris.

4. *Branches to the Lungs.*—The pulmonary plexus is made up of branches from the vagus and also from the sympathetic system, and it is difficult to say how much influence the vagus alone has on the lungs. It is certainly true that pressure on the pulmonary plexus is apt to cause septic pneumonia; probably the sensory fibres of the plexus supplying the bronchi are largely responsible for the production of the act of coughing.

5. *Branches to the Stomach and Œsophagus.*—Paralysis of the Œsophageal branches causes great difficulty in swallowing, while spasm of Œsophageal muscles is a recognised feature both in hysteria and also in association with tumour or other organic stricture of the Œsophagus.

The vagus supplies both sensory and motor fibres to the stomach, and upon the discharge of their functions the process of digestion largely depends. It is a well-known fact that vomiting is apt to be produced by stimulation of branches of the vagus, sometimes in the stomach itself, although often in some other branch of the nerve. Gastralgia or cramp in the stomach, probably the result of irritation of the sensory fibres of the vagus, and the gastric crises in locomotor ataxia, are unquestionably due to a lesion of the vagus.

It is very difficult to determine to what extent the vagus has been paralysed, although reference to the different branches will help, and in an involvement of the nerve trunk all its branches are apt to suffer to some degree. The close association of the glosso-pharyngeal nucleus renders it improbable that the nucleus of the one could be affected without an involvement of the other,¹ and the hypoglossal nerve is closely related to these two nerves in the first part of its course.

¹ In the schematic diagram on page 563 a common sensory and a common motor nucleus is figured for the 9th and 10th nerves.

Treatment.—Little need be said with regard to treatment. Where laryngeal muscles are paralysed, an attempt should be made to discover the cause of the condition, and if it be remediable, the affected muscle may be treated by massage or stimulated by electricity. Generally both poles are applied, one on either side of the larynx, and either the galvanic or faradic current may be used. In cases where there is bilateral abductor paralysis, and if the cords do not speedily assume the cadaveric position from the coincident involvement of the adductor muscles of the larynx, tracheotomy must be performed.

(10) *The 11th Nerve, or the Spinal Accessory Nerve*

The accessory or medullary portion of the nerve supplies the laryngeal muscles, and the fibres leaving the medullary nucleus form the internal and smaller division of the nerve. The other nuclear cells are situated in the cervical cord, and the fibres from them form the larger and external portion of the nerve, and supply the sterno-mastoid and the trapezius muscles. The medullary nucleus is apt to be involved in bulbar paralysis, and the spinal nuclear centre may be affected in various cord affections.

(a) PARALYSIS.—The nerve may be damaged by tumours, fractures, and other injuries involving the cervical vertebræ and the region of the neck. Sometimes neuritis involves especially the spinal distribution of the nerve, and it is due most commonly to exposure to cold and wet.

Paralysis of the laryngeal fibres has already been described in connection with the vagus nerve. In paralysis of the fibres supplying the sterno-mastoid and part of the trapezius the patient is unable to turn the head towards the opposite side, and the head may be retained in a slightly twisted position. The involvement of the trapezius is not complete, but shoulder movements are affected, and shrugging of the shoulders is carried out with great difficulty. The shoulder droops, and, owing to the unantagonised action of the rhomboids and the levator anguli scapulae, the scapular angle is rotated inwards. When the arm is raised, the scapula moves as a whole, the trapezius failing to fix the bone so as to permit of leverage for the deltoid muscle. It is hardly necessary to add that as the

trapezius is only partially supplied by this nerve, the degree of paralysis will vary in different patients.

A bilateral paralysis sometimes occurs, more generally as the result of progressive muscular atrophy; if both sterno-mastoids are affected the head falls back, but if both trapezius muscles are also paralysed it falls forward, giving the typical picture of an advanced progressive muscular atrophy. In any case the movement of the head is very slight, and the movements of both arms are also interfered with. The affected muscles are often flabby and wasted, and the electrical examination shows the typical reaction of degeneration. It is probable that the soft palate is supplied by fibres of the internal or medullary division of the nerve, and where, as in bulbar paralysis, the nucleus is affected, the voice becomes nasal from paralysis of the soft palate and food is apt to pass down the nose.

The **Diagnosis** is usually fairly easy, and the **Prognosis** depends largely on the nature of the affection; in both bulbar paralysis and progressive muscular atrophy it is by no means favourable.

Treatment.—In rheumatic neuritis an attempt may be made by counter-irritation and electricity to aid in the restoration of function.

(b) **TORTICOLLIS. WRY-NECK.**—The muscles which produce this spasm are not solely those functionated by the 11th nerve, although the sterno-mastoid and trapezius are mainly responsible. There are two forms which require description.

1. *Congenital Torticollis.*—This is the result of shortening of the sterno-mastoid muscle on one side. It is noticed soon after birth, and was believed to be due to traction on the head. In any case the affected sterno-mastoid is shortened and atrophied, and it has been suggested that the condition is similar to the lesion in infantile paralysis, in which talipes of the foot so often occurs. Any lesion which causes contraction of the sterno-mastoid will produce this condition, whether occurring in childhood or later life. Tenotomy affords relief.

2. *Spasmodic Torticollis.*—This is an affection in which there are active tonic and clonic spasms of the sterno-mastoid and other neck muscles.

The **Etiology** of spasmodic torticollis is unsatisfactory. Blows on, and injuries to, the region of the neck, and exposure to cold, are amongst the reputed causes while the family

history of patients affected often includes epilepsy and other nervous affections.

Both tonic and clonic movements may be present in the same patient, but, as a rule, this is not so.

In the *tonic form* of spasmodic wry-neck, the sterno-mastoid on one side is contracted, and draws the occiput backwards towards the affected shoulder, rotating the face towards the healthy side and tilting the chin upwards. Where other muscles participate in the tonic spasm, they may modify somewhat the position of the head, and not infrequently the trapezius muscle so participates, drawing the head somewhat more downwards. There may be a certain amount of pain accompanying the tonic spasm. The affected muscles tend to hypertrophy.


The *clonic form* of spasmodic wry-neck is much more serious, and while the sterno-mastoid is generally involved and sometimes the trapezius, other muscles of the neck, such as the splenius and the platysma, may also be affected. These clonic movements may occur almost constantly, although they cease during sleep. They are increased by nervousness or excitement, and they are often accompanied by considerable pain. The clonic movements may extend to other muscles in the neighbourhood, adding greatly to the patient's discomfort.

The **Diagnosis** is easy and the **Prognosis** is only favourable as a rule in tonic cases, and only in certain of these is it possible to afford relief. In a very few of the clonic cases the movements cease in course of time.

The **Treatment** consists partly in an effort to soothe spasms by sedative remedies such as the bromides, chloral, and even morphia, and partly, in severe tonic cases and with doubt of benefit, in dividing the external or spinal portion of the spinal accessory. In cases of clonic wry-neck, treatment is often useless, and division of the spinal accessory nerve rarely causes much relief.

(11) *The 12th Nerve, or the Hypoglossal Nerve*

This is the motor nerve for the tongue and for the muscles which protrude the tongue, and which are attached to the hyoid bone.

(a) PARALYSIS.—**Etiology**.—A lesion of the nucleus 

not uncommon in bulbar paralysis, and may be the result of encephalitis, sclerosis, or hæmorrhage. Where the lesion is supranuclear, the nerve fibres may be involved along with the motor fibres for the corresponding half of the body in the region of the internal capsule, or in the cortical centre of the tongue. The nerve may be affected by meningitis and

(*Private Proof.*)

PULMONARY EMPHYSEMA.

The pathological term for abnormal dilatation of pulmonary alveoli and air vesicles.

Etiology.—Secondary in many cases to bronchitis, is probably sometimes a senile change, and may be induced by occupations entailing excessive and continued strain on the lungs.

Clinical Features.—Cough if bronchitis, dyspnœa specially on exertion, later may be orthopnœa and even constant use of extraordinary muscles of respiration.

Chest shows increased antero-posterior measurement (barrel-shaped); upper ribs closer, lower ribs wider apart than normal, very obtuse epigastric angle; hyper-resonant percussion; prolongation of expiratory murmur; diminished vocal resonance; and displacement of neighbouring organs. Generally results in dilatation of right heart with consequent phenomena.

Pathological Anatomy.—Lung affected is typically bloodless, air vesicles running together, atrophy of walls and obliteration of capillaries. Anterior margins of lungs suffer most, but compensatory emphysema may occur in any part of lung.

Prognosis.—Is generally progressive, and if so leads to ever-increasing interference with circulation.

(1) PACHYMENINGITIS

INFLAMMATION of the dura mater.

(a) PACHYMENINGITIS EXTERNA.—This variety of pachymeningitis may occur where there is a fracture of the skull or

any neighbouring infective focus from which the outer surface of the dura becomes involved. It follows middle-ear disease and sometimes caries of bone.

As regards the spinal meninges, external ~~pochymanitis~~ may occur as the result of tuberculosis, sometimes from malignant tumours of bone, and more rarely from ~~actinism~~.

The lesion may be limited to the outside of the ureter or it may spread to the inner surface, and the clinical picture in most cases are those of irritation and later obstruction.

(3) **PACHYMENINGITIS INTERNA**—This may be secondary to external inflammation of the dura mater.

The most important variety of internal ~~polymerization~~ is unquestionably the form which gives the ~~special~~ special ~~kind~~ kind of ~~technological~~ technological ~~internal~~ internal ~~form~~ form ~~which~~ which ~~is~~ is ~~the~~ the ~~most~~ most ~~important~~ important ~~variety~~ variety ~~of~~ of ~~internal~~ internal ~~polymerization~~ polymerization.

Pachymeningitis Interna Hæmorrhagica—It consists in the formation of a false membrane on the inner surface of the dura mater, into which, sometimes, repeated hæmorrhages may take place in the substance of the dura.

Ecology.—This species is found in certain forms of country as the result of elevation and other conditions, such as particular soil and climate, but is well adapted together for all in which its subspecies is more or less abundant.

Pathological Anatomy.—The first point that a vascular neoplasm is formed in the first instance, and which is

3. **Clinical Features are variable.** There may be 1 or more episodes of nocturnal twitches and irritative attacks that be passed off early in the first year of age and which may be considered as the usual evidence of the condition. In most cases attacks and minor convulsions

[illegible]

ing changes in the pyramidal tracts, spastic phenomena may develop. The disease runs a chronic course, and may resemble in some respects syringomyelia.

(2) LEPTOMENINGITIS. ACUTE LEPTOMENINGITIS

Inflammation of the pia-arachnoid.

Etiology.—There are several special forms of meningitis, such as epidemic cerebro-spinal meningitis, referred to under infective fevers, and tubercular meningitis, to which reference will be made separately; but in addition to these, meningitis may result from many causes—

(1) It may be secondary to fracture or caries of the bones of the skull, and this includes a number of cases of middle-ear disease in which caries of bone has occurred.

(2) In cases in which there is a neighbouring focus of infection, as in middle-ear disease, mastoid abscess, or inflammations of orbit or nose, the membranes may become secondarily affected without necessarily any caries of bone.

(3) In acute infective fevers, such as pneumonia, erysipelas, septicæmia, and more rarely influenza, typhoid, and small-pox, meningitis may occur.

(4) In pyæmia, infective organisms may be carried, directly or indirectly, to the meninges of the brain and cord.

(5) Not infrequently, as a terminal stage of certain chronic diseases, such as Bright's disease, heart disease, etc., there may be infection of the meninges by pus-producing organisms, although the exact mode of attack is not always clear.

Pathological Anatomy.—The meningitis may be basal or cortical, the type depending on the cause of the condition and the original focus of infection, if any. In some cases it may be unilateral, as when secondary to ear disease, and as a general rule, unless free communication is interrupted, the spinal meninges suffer along with the cerebral. The effusion is generally purulent. It is usually sub-arachnoid in position, and it may cause very serious pressure on the cortex of the brain.

Clinical Features.—Along with the clinical features associated with the primary disease, the symptoms suggestive of the meninges having become involved are the following:—High temperature with headache, sometimes great sensitiveness to light (photophobia) and sound, and often delirium, merging

eventually into coma. There is rigidity of muscles particularly those of the neck, and sometimes convulsions, with paralysis of certain muscles, and more especially those supplied by the cranial nerves, may be noted in cases in which the effusion is mostly basal. There is occasionally Cheyne-Stokes breathing and sometimes the *tache cérébrale*. Cerebral vomiting is an early symptom, and may be associated with retraction of the abdomen. Optic neuritis is distinctly rare. Constipation is generally described as being a typical feature and often certain areas of skin, especially on the neck and calves, become hyperæsthetic. The pupils may yield suggestive information. Irritation produces contraction, but later during the paralytic stage the pupils become widely dilated. The pulse and respirations are often irregular and abnormally slow considering the degree of temperature present. Kernig's sign is generally positive. It is carried out by flexing the leg or the thigh at right angles, the knee being bent, and then attempting to fully extend the leg. In health this can be easily done, but in cases of meningitis, spasm of the flexors prevents full extension. A leucocytosis is not uncommon, and lumbar puncture offers important evidence both of the presence of pus cells and of excess of albumin in the cerebro-spinal fluid. Occasionally cultures made from the fluid yield definite information.

Diagnosis.—Although cases differ greatly in degree and severity, the clinical features are fairly suggestive and typical while the evidence obtained by lumbar puncture is most important.

Prognosis.—Death is extremely common. In non-purulent cases there may be recovery, but this is rare.

Treatment.—The head should be shaved and an ice-bag placed over it, while local blood-letting, by leeching over the mastoid, is often of great value where the patient can stand the operation. Counter-irritation is certainly useful, and blisters to the back of the neck are commended. Any suggestion offered by lumbar puncture should be followed up, and in certain cases antistreptococcic serum might be administered either hypodermically or by the rectum. Sometimes trephining is warranted, but it is improbable that much benefit will result from such treatment. The bowels should be freely opened, and the temperature kept in check by

sponging. Opium is often necessary for the relief of pain, and the patient should be kept at absolute rest in a darkened room. In most cases the spinal meninges suffer secondarily.

(3) TUBERCULAR MENINGITIS

Etiology.—It occurs most commonly in children, and usually under the age of 17, although sometimes it develops in adults. It is associated with hereditary or acquired tuberculosis.

Pathological Anatomy.—The membranes at the base of the brain are infiltrated with a yellowish, gelatinous exudation. Tubercles, often microscopic in size, may be seen following the course of the vessels, and especially in the neighbourhood of the optic commissure and the Sylvian fissure. Frequently the lateral ventricles are greatly distended with turbid fluid,¹ and the convolutions are markedly flattened as a result of pressure. It is a special type of leptomeningitis.

The spinal meninges are also affected, although in some cases not quite to the same extent. There may be tubercle nodules in the choroid coat of the eyeballs, sometimes even involving the retina; a primary lesion, sometimes a gland in the abdomen, more frequently an involvement of the lungs or of the genito-urinary tract, may also be recognised.

Clinical Features.—There are often definite premonitory symptoms. The child becomes peevish and out of health, and the disease is perhaps specially liable to develop after a continued fever such as measles or whooping-cough.

During the *stage of irritation* many of the most typical phenomena develop, such as headache, which is intense, and causes the child to give utterance to a short, sharp cry, often called the hydrocephalic cry. There is cerebral vomiting and fever, the temperature sometimes falling towards morning with considerable sweating. The little patient is excessively restless, starting out of sleep, and muscular twitchings are very common. During this stage the pupils are contracted. Various spasms, especially of muscles supplied by intracranial nerves, may be noted, and, in particular, squinting, while the head is bent back, the child apparently boring the occiput into the pillow, and the abdomen is often markedly retracted. The

¹ A condition which used to be called *acute hydrocephalus*.

breathing and pulse are both irregular, especially the latter, and the *tache cérébrale* is almost invariably noted.

This stage of irritation is generally followed by a *stage of depression or paralysis*, during which an ocular squint may develop, but of paralytic origin; the patient tends to become comatose, and convulsive seizures occur from time to time. The pupils, no longer contracted, are widely dilated, and the child generally sinks into what is best described as the typhoid state. During this period the temperature remains high, and usually a leucocytosis, if not definitely present before, is noted now.

Diagnosis.—The eyes should be invariably examined for the presence of choroidal tubercles, but it must be remembered that only in 50 per cent of cases of acute generalised tuberculosis do they occur. The classic phenomena, such as the retraction of the head, ocular squint (either spasmodic or paralytic), retraction of the abdomen, and the *tache cérébrale*, are very suggestive, and not infrequently a tubercular family history and the discovery of the primary focus of disease greatly assist the physician.

Prognosis.—Occasionally a temporary improvement occurs, and the hopes of friends and of the physician may be raised, but too often this is followed by a relapse with more marked symptoms, and in every case a grave prognosis should be given. In only an isolated case does recovery occur, and post-mortem it is rare to find evidence of an old quiescent basal meningitis of tubercular origin.

Treatment.—Unfortunately little benefit is derived even from the most careful treatment. An ice-bag over the head is advantageous, and sometimes the use of counter-irritants, but in every case the patient experiences relief from being kept absolutely quiet and in a darkened room.

IV. DISEASES OF THE SPINAL CORD

(1) INTRODUCTORY

THE position of the great ascending and descending tracts of the cord are seen in the diagram, which shows a transverse section of the cord just at the lower part of the cervical

enlargement. The *anterior* or *direct pyramidal tract* usually terminates about the mid-dorsal region; in a small number of cases it may be absent altogether. The *crossed pyramidal tract* situated in the lateral columns extends downwards throughout the whole cord.

The posterior columns of the cord are divided into the postero-median and the postero-external columns, the former ending in the nucleus gracilis, and the latter in the nucleus

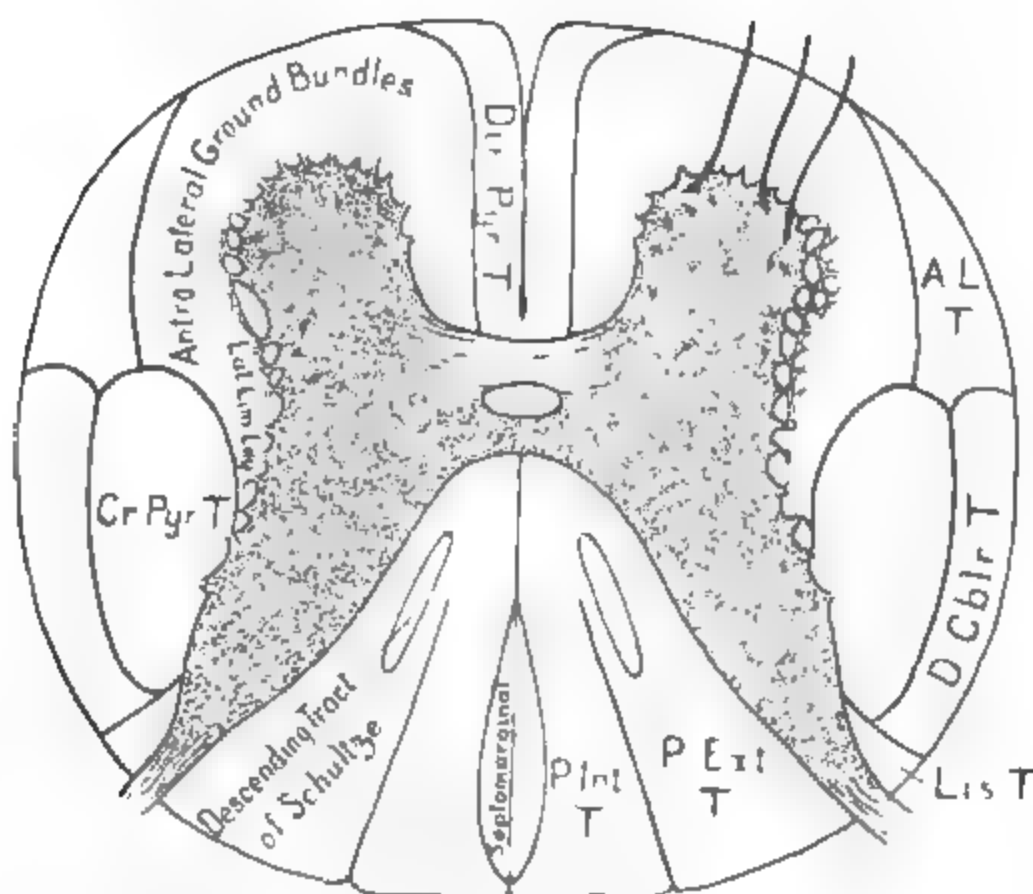


DIAGRAM 3d. — Diagrammatic Representation of Position of Tracts in the Spinal Cord. Dir. Pyr. T., Direct Pyramidal Tract. Cr Pyr. T., Crossed Pyramidal Tract. P. Int. T., Postero-Internal Tract. P. Ext. T., Postero-External Tract. D. Cblr. T., Direct Cerebellar Tract. Lat. Lim. Lay., Lateral Limiting Layer. Antro-Lateral Ground Bundles, Antero-Lateral Ground Bundles. A L T., Antero-Lateral Tract or Cumma-shaped Tract of Gowers.

cuneatus in the medulla. Fibres in the *postero-internal tract* pass up to the medulla, and mainly on the same side as that on which they enter, while most of the fibres in the *postero-external tract* leave the tract either to pass into the postero-internal tract, or to pass back into the grey matter of the posterior horn.

The *direct cerebellar tract* originates in the cells of Clarke's vesicular column, which can be traced upwards from the upper lumbar region of the cord, and the direct cerebellar tract begins in the lower dorsal region.

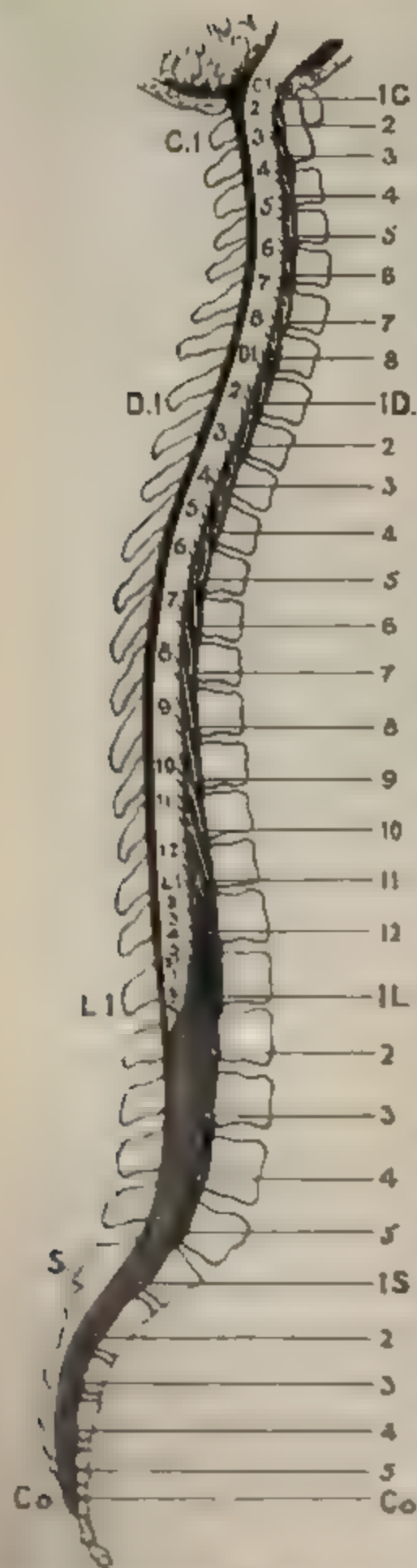


Diagram of the human spinal cord showing its segments and corresponding vertebrae. The diagram illustrates the relationship between the segments of the spinal cord and the corresponding vertebrae, showing that the cord does not extend as far down the spine as the vertebrae.

The *comma-shaped tract of Gowers* may be traced throughout the greater part of the cord.

The *tract of Lissauer* contains small-sized fibres which have entered at the posterior nerve roots, and which only run for a short distance in the tract, after which they pass upwards, mostly into the grey matter of the posterior portion of the horn.

The *small comma-shaped descending tract* in the postero-external column, the *antero-lateral descending tract* (not represented in the diagram), and the *septo-marginal tract*, do not call for description here.

In Diagram 37 the relationship of the segments of the cord to the spinal canal becomes clear and especially the fact that the cord does not extend much below the first lumbar spine so that in performing lumbar puncture a lower level is always selected. For a full description of the structure of the spinal cord reference must be made to special text-books. A brief account has already been given of the tracts carrying motor and reference has been made to the probable site of the tracts carrying sensory impulses in the cord.

2. BROWN-SEQUARD'S PARALYSIS

If a lesion of the cord is unilateral the following clinical features result—Just immedi-

ately above the level of the lesion there is a zone of cutaneous hyperæsthesia limited to the side of the lesion. In the affected segment or segments there is, in the first place, unilateral anæsthesia, and, in the second place, motor paralysis with unilateral wasting of muscles and the reaction of degeneration—in other words, the phenomena met with in a lesion of the second motor neurone. These sensory and motor changes are limited to the side of the lesion.

Below the lesion and on the *same* side as the lesion there is motor paralysis, but without wasting of muscles or electrical changes, because the lesion involves the crossed pyramidal tract, and is therefore a lesion of the first motor neurone. The tendon reflexes, after being in abeyance, become markedly increased. The Babinski sign is positive (see page 604). There is impairment of muscular sense, and often cutaneous hyperæsthesia, and the temperature of the affected limb is raised.

On the *opposite* side and below the level of the lesion the muscular power and tendon reflexes are normal. There is cutaneous anæsthesia, but muscular sense is normal, and the temperature of the limb is not raised. A unilateral lesion is more commonly the result of trauma, such as a gunshot wound, or similar injury, than of any disease.

The phenomena above described are well shown in the following diagrammatic plan :—

The Spinal Cord

| Zone of Cutaneous Hyperæsthesia. | | |
|--|-----------------|---|
| Paralysis of muscles, with wasting and electrical changes. Loss of reflex arcs. Zone of cutaneous anæsthesia. | Side of lesion. | Side unaffected. |
| Paralysis of muscles, but without wasting and electrical changes. Reflexes at first diminished, and later (in most cases) markedly increased. Babinski sign positive. Often cutaneous hyperæsthesia. Muscular sense impaired or lost. Temperature of skin raised. | | No paralysis of muscles. No interference with reflex arcs. Babinski sign negative. <i>Anæsthesia.</i> No loss of muscular sense. No change in temperature of skin. |

(3) PRIMARY SPASTIC PARALYSIS

(PRIMARY SPASTIC PARAPLEGIA—PRIMARY LATERAL SCLEROSIS)

A primary lesion of the pyramidal tracts, or of the first motor neurone, which might be termed idiopathic in contradistinction to cases in which the lesion is simply a descending degeneration.

Etiology.—It is not uncommon in patients suffering from influenza or syphilis, but it is difficult to differentiate between such cases and those in which the lesion is really secondary to a lesion of the tract at a higher level.

Pathological Anatomy.—There is sclerosis, and the sclerosis does not differ in any very obvious way from what is noted in secondary degeneration. It is probably the result of a toxin whose action seems to be limited to certain groups of fibres. Unquestionably the legs are more affected than the arms, and in certain cases the lesion extends to the trophic cells in the anterior horn of the cord, or may even involve the second motor neurone; in other words, the anterior nerve roots and their extension into the peripheral nerves may also suffer.

Clinical Features.—The chief symptom is weakness of the legs, which gradually increases, and in a matter of months interferes with the patient's ability to walk. When this occurs, he finds that he has inadequate control over movements which now become spastic in nature. He describes his legs as being stiff, and he tends to trip over any irregularities of the ground. The tendon reflexes are markedly exaggerated, and the Babinski test is positive. In this test, on tickling the sole of the foot, the toes separate and, together with the great toe, are dorsiflexed in place of being flexed into the sole as they are in health.¹ A lesion of the crossed pyramidal tract is always accompanied by a positive Babinski test, but the converse does not always hold true. Ankle-clonus is almost invariably present, and there is commonly the clasp-knife rigidity, by which is meant that when the flexed leg is partially extended, the muscles complete the extension in a sudden and spasmodic fashion. Spasm of the adductor muscles of the thighs is not uncommon. There are no sensory changes, excepting sometimes pains in the joints. Rarely does primary

¹ The dorsiflexion of the great toe is more readily and commonly seen in a positive test than separation of the toes.

optic atrophy develop, and there is no inco-ordination as a general rule.

Towards the final stages of the case, if a measure of quiescence is not established, there is a tendency to contracture of the affected limbs, while the organic reflexes fail and may cause much distress to the patient. Sometimes, though very rarely, the evidence of a primary spinal lesion is suggested by the existence of a girdle sensation.

Diagnosis.—There is no difficulty in arriving at the diagnosis that the pyramidal tracts are affected, but it is by no means easy to prove that the lesion is primary. The absence of inco-ordination prevents confusion of this disease with *ataxic paraplegia*.

The **Prognosis** is unfavourable, although partial recovery, or at least a quiescent stage, may be reached, during which the patient remains for a long time in the same condition, and the duration of life is not infrequently prolonged for years.

Treatment.—Drugs are unsatisfactory. In every recent case place the patient at absolute rest, and endeavour to keep up muscular nutrition by massage and electricity. Where there is even a suspicion of syphilis, administer potassium iodide and mercury. Sometimes sedatives are needful in order to relieve the spasmodic movements, and especially in cases where contracture has occurred.

(4) INFANTILE PARALYSIS

(ACUTE ANTERIOR POLIO-MYELITIS—ATROPHIC SPINAL PARALYSIS)

An acute, subacute, or rarely chronic inflammation of the anterior horn of the cord, the cell centre of the second motor neurone, but often selecting a limited area, or even a group or part of a group of multipolar cells. It is characterised by wasting of the muscles whose cell centres are involved.

Etiology.—Much difference of opinion has arisen as to whether the disease is infective, whether it is toxic, the toxin showing a peculiar selection of cells, or whether it may not be due to thrombosis or embolism of certain of the branches of the anterior spinal artery. It is common in childhood, and most generally during the first three years of life. Boys suffer more frequently than girls, and there are many more cases

in summer than in winter. There seems to be no doubt that definite epidemics have occurred, and if so, this favours the theory of an infective organism. The oft-repeated statement that a careless nurse letting the child fall was responsible for the illness, is probably inaccurate. The disease may attack children otherwise in perfect health, and it has occurred in adults, but in them it is by no means so common or so definite.

Pathological Anatomy.—The lesion consists of a limited acute myelitis, and there may be hæmorrhages at the chief foci of inflammation. Cases vary as regards the acuteness of the process, but there is always a resultant interstitial sclerosis secondary to the inflammation. The nerve cells in the affected area undergo very marked changes; in the first instance they swell up and appear to lose their processes; later they atrophy and may entirely disappear, or only a very few may remain in the area attacked. In slighter cases swelling of the nerve cells occurs, but they do not atrophy. The anterior nerve roots in a marked case become smaller, proportionate to the involvement of their cell centres, and there is descending degeneration in the anterior roots and the nerve fibres. The muscles supplied by these nerve fibres atrophy, and undergo fatty degeneration. It is doubtful whether the actual inflammatory process passes down the anterior nerve roots; so far as we are aware at present, the changes found in the nerves and muscles are secondary to the primary change in the anterior horn.

The enlargements of the cord, either cervical or lumbar, are the common sites of the foci of inflammation in the anterior horn. The disease may be unilateral or bilateral, sometimes both arm centres suffer more or less completely, sometimes an arm and a leg on the same side, or on different sides, and, as already indicated, the extent of inflammation is often limited to groups of muscles.

As development goes on, and should recovery not occur, the affected anterior nerve roots remain atrophied, and the corresponding half of the cord is seen to be smaller than its fellow.

Clinical Features.—Marked constitutional disturbance, with malaise, vomiting, and fever, indicates the onset of the attack, which may be extremely sudden. When the acute symptoms have passed off, the paralysis is noticed. The paralysis is usually wide-spread to begin with, but the

majority of the affected muscles or groups of muscles rapidly improve, whilst a limb or part of a limb remains paralysed. The muscles permanently affected rapidly waste, and the reaction of degeneration is readily obtained; where a limb remains paralysed, its growth and the development of its muscles are greatly interfered with, thus causing many familiar deformities, such as club foot, which result from the contracture and unresisted action of non-paralysed or less paralysed muscles. Pain, which may be referred to the part about to be paralysed, is sometimes an initial symptom; after the paralysis has developed, local tenderness may persist. It is probably of neuritic origin, but there are no other sensory changes. In very wide-spread cases all four limbs may be paralysed, and it is even possible to have paralysis of trunk muscles, the disease eventually proving fatal. As the lumbar enlargement is the centre for the organic reflexes governing the bladder and bowels, we occasionally find incontinence of urine and feces.

In the arm there may be an upper-arm type of paralysis which includes the deltoid, biceps, brachialis anticus and supinator longus, or a lower-arm type of paralysis, which includes the extensors and flexors of the fingers and wrists.

In the legs the tibialis anticus and the extensor muscles are more generally affected than the hamstrings and gluteal muscles. The muscles of the face are rarely involved.

To the touch the affected muscles are flabby and soft, and the reaction of degeneration (page 538) should be obtained from 7-10 days after the attack commences, although in a favourable case, in which there is a prospect of recovery, only a partial reaction of degeneration is present. The superficial and deep reflexes of the affected limbs are generally absent. In cases where a whole limb is affected the growth of the limb is greatly interfered with, and a baby arm or a baby leg may result, often greatly handicapping the patient throughout life. There is no resultant change in the joints excepting the fact that bones may not be kept in their positions when the supporting muscles are completely paralysed, and subluxation sometimes occurs.

The **Diagnosis** presents no difficulty. The history of the case, with the age of the patient, the absence of sensory phenomena, and the presence of paralysis, and, lastly, the

electrical reactions prevent any possible error. In *peripheral neuritis* pain is typical, and pain felt both in the muscles and nerves; in infantile paralysis pain may occur, although it is rare, but it does not persist for any length of time, and then peripheral neuritis is by no means a common disease in childhood. It is almost incredible that *hip-joint disease* could be mistaken for infantile paralysis.

Prognosis.—While it is hardly likely that even the smallest focus of inflammation can disappear without leaving some trace, a great diminution in the extent of the paralysis may be hoped for in most cases, and the electrical reactions should suggest a favourable prognosis, especially where an incomplete reaction of degeneration is present.

Treatment.—Give a purge and a fever mixture, should the case be seen in its early stages. Keep the patient absolutely at rest, and wrap the affected limb in cotton wool. It is unwise to apply fly-blisters or other severe counter-irritation to the region of the spine, because, although bedsores are by no means common, no benefit has been found to follow this procedure. As soon as possible endeavour to keep up the nutrition of the affected muscles, and do this by massage and electricity. Where there is an incomplete reaction of degeneration, the affected muscles may be stimulated by the faradic current, and such treatment is most efficacious. Massage and electrical treatment should be kept up for months, and improvement may be hoped for during the whole of that time. In the later stages, when the chance of benefit from massage and electricity is lessened, endeavour to prevent deformity: much may be done by artificial supports for strengthening the limb, while transplantation of tendons, so as to make flexor muscles take the place of paralysed extensors, has yielded results which, if not brilliant, are at least extremely good.

There are few medicinal agents likely to be of much value. certainly strychnine in small doses is worth trying when the acute inflammation stage in the cord has subsided. ergot and belladonna amongst other remedies have been administered with benefit in certain cases during the early stages. Too much, however, should not be expected from the use of any internal drug.

Paralysis in Adults.—There is an acute form of

this disease affecting adults just as it affects children, and usually beginning with a sharp febrile attack, but it is extremely uncommon.

The chronic form of the disease is said to be peculiar to adults, but little can be said definitely about a lesion which is so rare.

(5) ACUTE ASCENDING PARALYSIS

(LANDRY'S PARALYSIS)

This is a rapidly developing motor paralysis, travelling from below upwards, and usually fatal.

Etiology.—Cold and wet, syphilis, and possibly alcoholism, and such toxins as those of diphtheria and typhoid, have been considered as causes. Males suffer more than females, and it is generally a disease of adult life.

Pathological Anatomy.—Changes have been described in the grey matter of the anterior cornu, but many authorities consider that no naked-eye lesion is present. Some believe that it is an ascending peripheral neuritis. It may be an affection, toxic in nature, of the whole lower motor neurones.

Clinical Features.—After some premonitory tingling, paralysis with loss of reflexes commences, travelling from the legs upwards. The arms suffer less completely than the legs, and with involvement of the other muscles, difficulty in respiration, deglutition, and speech are observed. The affected muscles do not waste, nor show the reaction of degeneration, save in exceptional cases. The organic reflexes, sensation, and the trophic, vasomotor, and mental functions are normal as a rule. The spleen is frequently enlarged, and profuse sweating is common, but there is no pyrexia.

Diagnosis.—The advancing paralysis of muscles without pain renders the diagnosis easy.

Prognosis.—It rarely terminates in recovery, generally in extension to the medulla, and death follows in 2 days to 2 weeks.

Treatment.—Hot baths may be tried at first, and also counter-irritation to the back. No drugs are of much value. Give ergot, strychnine, and perhaps salicylates, especially if the condition is due to cold and wet. Quinine and per-

chloride of iron may also be administered where there is any probability of the case being of septicæmic nature.

(6) PROGRESSIVE MUSCULAR ATROPHY¹

A lesion involving motor nerve cells in the anterior horn of the cord, probably the result of a toxin and characterised by progressive wasting, with fibrillary tremors in the muscles affected. It is a lesion, therefore, of the second motor neurone, although it seems probable that the disease may spread upwards to the first motor neurone in certain cases.

Etiology.—It is commoner in males, and generally between the ages of 25 and 45. It is certainly the result of overwork, exposure, and worry, and there is a hereditary tendency to this disease strongly marked in certain families. A rare form, occurring in infancy, appears to possess this hereditary characteristic.

Pathological Anatomy.—The ganglion cells in the anterior horn of the cord show the most marked changes. Certain of these become atrophied, and may even disappear, while the grey processes are less numerous, and the chromatic substance largely decreases in the affected cells. Increase of neuroglia takes the place of the cells which have become atrophied, and the anterior nerve roots in the affected segments of the cord are wasted, while the muscles supplied by the affected cells are pale and atrophied, and show indistinct striation, and often fatty, more rarely hyaline, degeneration.

In a special group of cases the pyramidal tracts are also involved, giving rise to spastic phenomena along with the atypical wasting of certain groups of muscles. In not a few cases, before death, the toxin affects nuclei in the medulla, and may therefore lead to the development of bulbar paralysis.

Clinical Features.—Rheumatic pains are often the earliest clinical feature, and later one or other type of muscular wasting develops.

I. DUCHENNE-ARAN TYPE.—The disease generally begins in the arms, and the thenar and hypothenar eminences disappear, the interossei and lumbricales waste, and as a result of shortening of the extensors unresisted by the interossei, the griffin-like hand, called by Duchenne the *main en griffe*, is

¹ Compare Idiopathic Muscular Atrophy, p. 729.

produced. Usually one hand is first affected, and the wasting of the deltoid or biceps, and the flexors, followed later by the extensors, soon shows that the disease is involving the muscles in the upper part of the arm. Later the lower two-thirds of the trapezius and certain of the scapular muscles waste. Muscles of the trunk may follow: those of the neck and legs, if affected at all, generally succumbing at a late period. In all the muscles affected, fibrillary tremor or twitching is very common, there is loss of electrical irritability, and there may be a partial reaction of degeneration. The tendon reflexes are gradually lost, but sensation is unaffected and the organic reflexes are normal.

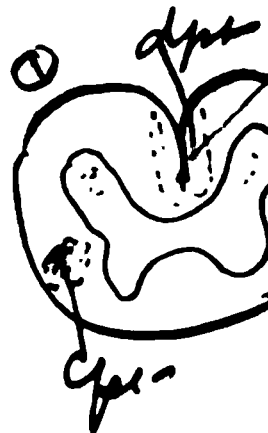
II. THE PERONEAL TYPE.—This is a less common form of progressive muscular atrophy, in which the peronei and anterior tibial muscles of the leg are first and most markedly affected. Double talipes varus may develop, and as the case progresses, the arms and the body generally become involved.

Where, with wasting of the arm and shoulder muscles, the crossed pyramidal tract has also suffered, causing spastic paralysis of the leg, the condition has been termed *amyotrophic lateral sclerosis*.

Diagnosis.—The history of the case, and the involvement of the interossei, the lumbricales, and the thenar and hypothenar eminences, are typical of the disease, and the absence of sensory phenomena prevents the possibility of mistaking progressive muscular atrophy for *syringomyelia*. In *paralysis of the ulnar nerve* the whole of the interossei are not involved, and therefore it is easy to differentiate between the griffin-like hand of ulnar paralysis and that of this disease.

The **Prognosis** is unfavourable, although the disease may become arrested at any period.

The **Treatment** is most unsatisfactory; strychnine and arsenic, and certainly antisyphilitic remedies, should be tried. Probably strychnine is most efficacious when injected hypodermically into the affected muscles. Electricity, both galvanic and faradic, and massage are useful in preventing more rapid wasting of muscles. Attention should be paid to the feeding and general comfort of the patient, and as far as possible the deformity due to the action of muscles not antagonised should be prevented.



(7) LOCOMOTOR ATAXIA—TABES DORSALIS

A disease due to a degeneration of the posterior roots and part of the posterior columns of the cord; characterised by marked inco-ordination, loss of knee-jerks, shooting pains, and many other symptoms and signs indicative of similar degenerative changes in other parts of the nervous system.

Etiology.—The disease is most common in males, and after middle life. It is probably the result, in a large percentage of cases, of syphilis, although this is not definitely proved. Concussion of the spine, over-exertion, damp or cold, and alcoholic excess, have also been included as possible causes. Recently it has been suggested that the disease may have its origin either in defective (an embryonic error) cord tracts upon which the strain of life has begun to tell, or from the excessive use of certain tracts which for a long time had not been called upon to transmit so many stimuli.

Pathological Anatomy and Pathology.—The posterior columns of the cord present the greatest changes. The sclerosis is most extensive in these columns in the lumbar region, and it diminishes as the cord is followed upwards. Above the lumbar enlargement the postero-external columns become gradually less and less affected, but the degree of involvement of these columns varies considerably in different cases. It is probable that the sclerosis commences in the posterior root zone in the lumbar region of the cord, and above that level of the cord the posterior roots are generally healthy. In cases where the arms are markedly ataxic, the sclerosis of the posterior roots is also present in the cervical enlargement of the cord, and associated with this is a profound sclerosis of the postero-external tracts generally throughout the entire length of the cord. The anterior part of the postero-external tracts, even in the lumbar region of the cord, usually escapes sclerosis. In the postero-internal tracts the fibres which enter these columns at different levels of the cord do so anteriorly, that is, next the commissure; and, therefore, in tracing the postero-internal tracts upwards in an ordinary case of locomotor ataxia, in which the lumbar enlargement presents the most marked sclerosis, we find that the anterior part of these tracts contains healthy fibres which have entered by healthy nerve roots.

If we examine carefully the posterior cornua, there are indications that the sclerosis begins in the posterior nerve-root fibres, and that the tract of Lissauer almost invariably suffers, at least for a certain distance above the lumbar enlargement. It seems probable that, as Marie states, the lesions of the spinal cord in locomotor ataxia occur by segments, and therefore where degenerated nerve-root fibres cease to enter the cord, tracts which are not continuous to the medulla will be found to be healthy. Following the posterior nerve-root fibres backwards, we may find the ganglia affected, although this is not constantly the case, and sometimes a neuritis is present, extending down along the nerves.

Many other tracts and parts of the cord may suffer; sometimes the cells of Clarke's vesicular column are degenerated, and we note that the direct cerebellar tracts which, according to many authorities, originate in these cells, are sclerosed. The comma-shaped tracts of Gowers, occasionally the pyramidal tracts, and sometimes the anterior cornual cells, may also be involved.

In a proportion of cases there is chronic meningitis, and it has even been suggested that the disease may originate in connection with the pia mater, although this is unlikely.

Microscopically the nerve fibres in the degenerated tracts are markedly atrophied, and, as the name sclerosis implies, there is great increase of connective tissue.

Certain of the cranial nerves and special bundles of nerve fibres in the brain may undergo degeneration. In this way the optic nerves may be affected by a primary degeneration, and the 3rd, 4th, 6th, and sometimes other cranial nerves have been found involved. The connections of the 5th nerve with the cord and the sympathetic fibres which supply the dilator pupillæ muscle are often affected, particularly the latter.

In a small proportion of cases of the disease there is a marked change in the bones and joints. The articular ends of some of the long bones, and especially the femur, undergo atrophy, while the joints swell up and there may be considerable disorganisation. New bone formation may develop around the joint much like the condition in osteoid arthritis, and fracture of long bones may occur owing to their excessive brittleness.

The pathology has been so far discussed both under

etiology and in connection with the pathological anatomy. Whether the sclerosis results from a toxin or not we cannot as yet say, but the disease is one affecting the sensory nervous system, although the initial site of lesion is inextricably mixed up with secondary degenerations. It seems certain that anti-syphilitic treatment, although too often a failure, has yielded better results than any other method.

Clinical Features.—There are three more or less distinct stages of the disease.

Stage 1.—Here we have commencing inco-ordination, numbness of the skin of the feet, shooting pains, especially in the legs and round the trunk in the line of the spinal nerves, loss of knee-jerks, the presence of the Argyll Robertson phenomenon (pupil reacting to accommodation, but not to light), sometimes transient paralysis of an eye muscle, and sometimes incontinence of urine. A pre-ataxic stage has also been differentiated, but it seems an unnecessary subdivision.

Stage 2.—In this stage we have more advanced ataxic gait, increased pains, myosis (contracted pupil), impotence, girdle sensation, sometimes deafness, and occasionally curious crises—laryngeal, gastric, vesical, and so forth.

Stage 3.—Ataxia and anæsthesia become still more marked, there is often ophthalmoplegia, and as time goes on the organic reflexes may give way, the patient becoming increasingly helpless and burdensome both to himself and to those about him.

Where trophic changes in the joints begin in the first, Charcot's disease results in the third stage.

The following is a brief description of the typical phenomena noted under their appropriate headings:—

(1) *Sensory Phenomena.*—The *shooting* or *lightning* pains are very typical. They come on at night, occur in the legs, and sometimes the trunk, are apt to be paroxysmal, and may last for hours and even days in one position, and may then change to some other part of the body. Not infrequently the pains are modified by the weather, becoming worse during cold and damp, while with fatigue or indisposition their severity increases.

The *girdle sensation* of locomotor ataxia is a feeling of constriction resembling that produced by a tight belt, and its level is probably associated with the diseased segments. *Paræsthesia* or abnormal sensations of different kinds

common, such as burning, pricking, numbness, and formication; they are apt to be felt in the feet, and less frequently in the hands. There is anæsthesia over areas in the feet and legs, and generally, even when sensations are appreciated, their conduction is delayed. In a few cases *polyæsthesia* is present, by which is meant that one touch is felt by the patient as many, and also *allocheiria*, which signifies that a touch on one side of the body may be referred to the same position on the opposite side.

In some cases, so insensible does the skin become that a perforating ulcer on the sole of the foot occasions no pain to the patient, and the muscles and bones may be equally anæsthetic.

Muscular sense is sometimes *abolished*, but by no means always so. It can be tested by investigating the ability of the patient to recognise different weights lifted, but care must be taken that the weights are fixed to the legs in such a way that the pressure upon the skin does not convey to the patient the information which it is desired he should gain by the amount of muscular effort put forth. The bag containing the weight should, therefore, be fastened to the limb tightly. Another method of testing muscular sense is by inquiring of the patient, with his eyes shut, where his leg has been placed, and getting him to point to the position of the great toe of the foot which has been moved.

(2) *Reflexes*.—The knee-jerks are always abolished, and in most cases the other important muscle reflexes in the legs are found to be absent or very feeble. Where the arms are affected, the wrist-jerk, biceps-jerk, and other tendon reflexes will also be abolished.

The skin reflexes of the legs are naturally lessened in any case owing to the degree of anæsthesia usually present.

(3) *Motor Symptoms*.—The leading phenomenon is *ataxia* or *inco-ordination*, and this in most cases (where the cervical enlargement of the cord is not involved) is limited to the legs. Romberg's phenomenon is well marked, and may, in fact, be the first indication to the patient that something is wrong. When he stands, with heels together and eyes shut, he sways and tends to fall, and when washing in the morning or soaping the face he may reel against the wall or knock over the basin.

Romberg noted that by this test the inco-ordination, which

develops gradually, is easily recognised. The gait is soon modified, as the result mainly of the ataxia. The alteration becomes more observable when the patient endeavours to walk without boots or 'shoes, and it is seen that he separates the legs so as to give himself a wider base of support. He raises his feet too high, he brings them down, heel first, with a thump on the floor, and it is found that when an angled chalk-line is drawn on the floor he cannot with the great toe trace it accurately. Notwithstanding these changes there is no muscular weakness, as a general rule, and the power of the patient may be unaffected.

In cases where the cervical part of the cord is involved, the arms also become inco-ordinate, and in rare cases they may show inco-ordination prior to its development in the legs. The inco-ordination in the arms may be well demonstrated by making the patient, with eyes closed, endeavour to touch the tip of the nose with the forefinger, or to bring the tips of the fingers of the two hands into apposition.

(4) *Special Senses*.—The *Argyll Robertson phenomenon*, by which is meant the loss of pupil reaction (contraction of pupil) when the eyes are exposed to the light after being closed, is present in 5 out of every 6 cases of the disease, if not in an even larger proportion. The phenomenon depends on degeneration of Meynert's fibres, which join the anterior corpus quadrigeminum to that part of the nucleus of the 3rd nerve governing the sphincter pupillæ muscle. It is also found that pinching the skin of the neck no longer dilates the pupil, and that, owing to the involvement of the sympathetic fibres in the cord which supply the dilator pupillæ, there is generally persistent contraction of the pupil termed *spinal myosis*.

Primary atrophy of the optic nerve is certainly not common, and probably is not present in more than 1 out of 10 cases. It begins during the first stage, and with the advent of complete blindness there is often an apparent arrest of the disease in other directions. Certain of the *external ocular muscles* may be temporarily *paralysed*, and of these the levator palpebræ not very infrequently suffers, causing ptosis to develop, while sometimes the ciliary muscle may also be affected. In later stages of the disease a permanent paralysis of one or other of these oculo-motor muscles may develop. *Deafness* and *loss of the*

sense of smell (anosmia) are sometimes present. Associated with the laryngeal type of crisis there may be a degree of paralysis of the abductor muscles of the glottis.

(5) *Sphincters*.—The bladder gives trouble at an early period in the disease. There may be *incontinence of urine*, sometimes retention, sometimes mere sluggish action, and owing to the cutaneous anæsthesia the patient may be ignorant of nature's calls. In some cases cystitis ensues during the later stages of the disease.

The bowels may give similar trouble, the sphincter becoming weak, and should diarrhœa be present, much trouble is caused to the patient from *incontinence of fæces*. Fortunately constipation is common, and it is remarkable in how many cases, after control of the sphincter has been lost, it may be temporarily or permanently regained.

(6) *Vasomotor and Trophic Functions*.—There is sometimes *local sweating*, sometimes alteration in the distribution of pigment in the skin and hair. The *perforating ulcer* of the foot has already been mentioned; it usually originates in a corn on the sole of the foot. The teeth may drop out, the bones, especially the long bones, may become brittle and their articular ends may be partially absorbed, while the joint changes, which perhaps most commonly affect the knees, and which go by the name of *Charcot's joint disease*, develop during the third stage of locomotor ataxia. The resulting disorganisation of the joint often renders any hope of treatment absolutely futile, and even in the best conditions there may be new bone thrown out, so that ankylosis and deformity inevitably ensue.

(7) *The Crises* are not common; in the *gastric* variety there is severe pain in the stomach, accompanied by vomiting of a watery fluid which may contain bile or blood, and often by severe hiccough. *Rectal* crises imply terrible tenesmus, and *urethral* crises mean similar pain at the neck of the bladder. *Laryngeal* crises are simply laryngeal spasms, which may give trouble and frighten the patient by the feeling of impending suffocation.

Complications.—It is only necessary to glance at the pathological anatomy in order to realise the many complications of the nervous system which may ensue, due to the spread of the sclerosis or to a myelitis being set up in some part of the cord. Myelitis, acute and chronic, sclerosis of

lateral columns of the cord, progressive muscular atrophy, and general paralysis of the insane give some indication of complications which may render the diagnosis difficult.

It is also probable that other systems may be affected, and cardiac disease is not uncommon in cases of locomotor ataxia.

Diagnosis.—A typical case is easy, and the absence of knee-jerks, the presence of the Argyll Robertson phenomenon, the ataxia and the pains, present a clinical picture different from any other cord lesion. Attention is directed to the differential diagnosis of *ataxia paraplegia* and *Friedreich's ataxia* (see pp. 622 and 620), while in *cerebellar tumour* the presence of optic neuritis is practically constant.

Prognosis.—At an early stage in the disease, and especially if the condition be of syphilitic origin, there is hope of improvement from antisyphilitic treatment. As already stated, the presence of optic atrophy, with the inevitable blindness to which it dooms the patient, generally precedes the arrest of the disease otherwise.

The first stage may last for twenty years, or the disease may progress with much greater rapidity, at times becoming stationary and again advancing. Complications not infrequently are the cause of death, and should the patient become bed-ridden, bedsores are to be dreaded, and bladder troubles may result in kidney disease.

Treatment.—Invariably try antisyphilitic remedies carefully and patiently; of these potassium iodide will probably yield most benefit. Forbid all mental and bodily fatigue, and keep in mind that a period of rest in bed is often of great service. In mild cases recommend a sea-voyage, and regulate the patient's life, and especially the amount of alcohol and tobacco which he should use. Caution the patient against sexual excess during the early stages of the disease. In the treatment of symptoms remember that in certain cases nothing affords so much relief for the pains as electricity, sometimes galvanism and sometimes high-frequency, while hot baths and a change to a warmer and drier climate are helpful. In some cases it is necessary to give hypodermic injections of cocaine or morphia. For the crises the nitrite group, and especially nitrite of amyl and nitro-glycerine, give great relief. Antipyrine, phenacetin, and kindred remedies are beneficial in arresting the pains of tabes.

Locally we should try counter-irritation along the line of the spines, and especially in the neighbourhood of the affected cord segments. Iodine and blisters of different kinds are good, while hydrotherapy has certainly yielded beneficial results in a few cases.

For the disease itself there is no remedy which can honestly be called a specific. Arsenic, silver nitrate, quinine, strychnine, phosphorus, Calabar bean, ergot, belladonna, and many other drugs have been administered, but they are all unsatisfactory. Pay special attention to the bladder and never permit retention to occur, and guard very carefully by antiseptic precautions against the risk of inducing a cystitis by passing a dirty catheter. It is wise also to warn the patient of the possibility of a perforating ulcer of the foot, and with this object in view recommend attention to any corn or injury, and prevent the patient from attempting to cut or pare corns himself.

Two other methods of treatment demand a word: one is the outcome of the suggestion made some years ago to treat locomotor ataxia by stretching the spinal column, and with it the spinal cord. This has been done either by suspending the patient practically from his head, or with the patient lying on a couch, by bending the back in a bow-shaped fashion to bring the knees into as close contact with the face as the operator may consider advisable. The treatment has not yielded good results, and is certainly not free from risk.

It has, secondly, been found possible to do something for the cure of the ataxia by re-educating the patient to perform movements in a co-ordinate way, and much may be accomplished by training him to walk along a straight line with the artificial help of a go-carriage.

(8) FRIEDREICH'S ATAXIA—HEREDITARY ATAXIA

A combined sclerosis of the spinal cord, involving both the posterior and lateral columns, and commencing at an early age. Professor Friedreich of Heidelberg first described the disease in 1861.

Etiology.—As the name implies, the disease is generally hereditary, several members of a family being, as a rule, affected, but direct transmission is rare. Alcoholism and syphilis have been traced in certain cases in the parents,

but the percentage is not convincing. The disease begins in early life, generally between the ages of 6 and 15, and very rarely after the age of 25. Males suffer more frequently than females.

Pathological Anatomy.—The sclerosis affects the posterior columns very markedly, and to a less extent the lateral and anterior columns. The posterior columns are extensively sclerosed throughout the entire cord, while the sclerosis only involves the periphery of the lateral and anterior columns, extending forward almost to reach the anterior commissure. This implies the involvement of the direct cerebellar tract (and associated with it atrophy of Clarke's vesicular column) and the antero-lateral tract of Gowers, while the pyramidal tracts are less extensively affected.

The pathology of the condition is by no means clear. It seems most probable that it is an embryonic error in development, owing to which the tracts affected cannot resist the strain of life. It is, however, difficult to assert that it is an early decay of these affected tracts. The microscopic appearance is that of proliferation of connective tissue.

Clinical Features.—As indicated under etiology the disease generally commences between the ages of 6 and 8, and comparatively rarely after the age of 15. The ataxia begins in the legs, but soon also develops in the arms, and in them becomes very marked. The gait is peculiarly affected, somewhat resembling in its swaying, irregular movements the walk of a drunk man. The movements of the arms, instead of being merely ataxic, have a choreiform and swaying character, not very unlike that of the legs. Romberg's phenomenon is well marked, and the knee-jerks are absent. The speech is peculiar, being somewhat slow, and suggesting sluggish cerebration, while nystagmus, and exceptionally the Argyll Robertson phenomenon, are present. There is often vertigo, but primary atrophy of the optic nerve does not occur. Trophic changes in the skin and joints, and incontinence of urine, are generally absent. As time goes on, certain deformities develop. These are lateral curvature of the spine, and very generally talipes equinus, the patient also tending to walk on the outer side of the foot.

Diagnosis.—The fact that it is a family disease, inasmuch as several members of the same generation are usually affected, is a great help in diagnosis, while the ataxia of the arms, and

the peculiar choreiform or jerky movements of both arms and legs, the affected speech and well-marked nystagmus prevent any confusion with *locomotor ataxia*. In addition the age of the patient is practically always under 25, while in locomotor ataxia, or *ataxic paraplegia*, the age is generally over 40. The gait may suggest *cerebellar tumour*, but optic neuritis is absent, and the other characteristic features of Friedreich's ataxia should prevent a mistake being made.

Prognosis.—The disease may last for many years, with gradual advance, and is generally fatal by some intercurrent affection. Unfortunately there is a tendency to various deformities and contractures, as already noted, and these may cripple the patient and much interfere with his comfort.

The **Treatment** is practically *nil*. Attention to the general health is of course desirable, and much may be done to obviate deformity and contracture by careful massage, and in the later stages by division of tendons.

CEREBELLAR HEREDITARY ATAXIA OF MARIE.—This form of ataxia starts in somewhat later life than Friedreich's disease, usually after the age of 20. There is marked ataxia, disordered speech, and nystagmus, but the knee-jerks are exaggerated; and there is no scoliosis. Little can be said as yet about this rare affection; the cerebellum is evidently involved, and the gait is more typically cerebellar than it is in Friedreich's ataxia.

(9) ATAXIC PARAPLEGIA

This is a combination of sclerosis of the posterior and lateral columns developing in later life, and so is distinct from Friedreich's ataxia, while there is less extensive involvement of the posterior columns than in that disease.

Etiology.—It is a disease of males, the ages being generally from 30 to 40, although females are not exempt. The cause of the condition is doubtful; syphilis, over-work, cold, injury to the spine, and sexual excesses have all been considered as possible predisposing or exciting causes.

Pathological Anatomy.—In the posterior columns the sclerosis is most marked in the dorsal region of the cord, while it is less intense in the lumbar region; the posterior root zone, which is so markedly affected in locomotor ataxia, escapes.

In the lateral columns, the crossed pyramidal tracts, Gowers' tract, the direct cerebellar tract, and the lateral limiting layer may all be involved, and are more sclerosed in the dorsal than in the lumbar region of the cord. The meninges of the cord do not appear to be affected.

Clinical Features.—It is a slow disease, the patient complaining, to begin with, merely of languor, and the history suggests ordinary spastic paralysis. The legs suffer first, and Romberg's phenomenon is marked; but the knee-jerks are exaggerated, and ankle clonus is present, because the root zones in the lumbar enlargement have escaped. There are no pains, there is no girdle sensation, and there is not the typical heel-thumping gait of locomotor ataxia. The arms may be ataxic, but there is usually no Argyll Robertson phenomenon, and rarely optic atrophy. There may be slight difficulty in speaking, with tremulous movement of the face, but these symptoms are not always present. The Babinski sign is generally positive.

As the disease advances, the patient becomes bed-ridden, the condition closely resembling the later stages of a severe case of spastic paralysis, and the organic reflexes may give way.

Diagnosis.—The association of ataxic and spastic phenomena marks the disease out sharply from *tubes*, while from *Friedreich's ataxia* it is equally sharply differentiated by the age of the patient, the exaggerated knee-jerks, and the absence of nystagmus, and of the typical movement superadded to the ataxia in the arms and legs in *Friedreich's ataxia*.

Prognosis.—The disease may last for years, and it is rarely arrested. Death is apt to be due to some intercurrent affection, and may very probably be associated with the bladder.

Treatment.—The use of hot baths and residence in a warm climate are useful in this disease, just as they are in spastic paralysis. In cases with a syphilitic history give potassium iodide, and possibly mercury. Do not use galvanism or faradism, and avoid the administration of strychnine; on the other hand, give arsenic, which is sometimes of benefit, and it is possible that hydropathic treatment may do partial good. Stretching the spinal column has been recommended for this disease, just as for locomotor ataxia, but there is a considerable element of risk, and the success attained by the treatment hardly warrants its trial.

(10) MYELITIS

An inflammation of the spinal cord, which may be acute, subacute, or chronic, and in extent may be diffuse or localised. Sometimes localised areas of myelitis are disseminated throughout the cord.

Etiology.—*Acute myelitis* may result from injury, hæmorrhage, tumour, caries, meningitis, exposure to cold, the effects of over-exertion, and, lastly, from various infective fevers, and especially small-pox and typhus, to which it may be a sequel.

Chronic myelitis may be the result of syphilis, possibly of alcoholism, although more probably these will merely be predisposing factors, and it may be due to any inflammatory condition, in which the original lesion causes a chronic in place of an acute inflammation.

Pathological Anatomy.—Both white and grey matter may be affected, although the grey matter suffers most severely. The nerve cells tend to swell up, lose their processes, and show chromatolysis, and in many instances eventually disappear, and there is frequently hæmorrhage associated with the area of inflammation. On section of the affected cord the substance is found to be diffuent and congested, and may in a severe case be suffused with blood. The nerve fibres are broken up, and corpora amylacea, which are probably simply myelin droplets, are often noted throughout the white matter of the cord. There may be distension of blood-vessels, an invasion of leucocytes, and eventually sclerotic changes, which take the place of damaged areas of the cord. Various terms have been applied indicating the appearance of the affected area of the cord. For example, *red* softening indicates the suffusion of the affected area with blood, *yellow* softening implies that the colour of the hæmorrhage once present has faded, and *white* softening indicates that no congestion can be made out. The nerve roots may be secondarily involved, and not infrequently the meninges show definite meningitis, which may, in fact, be the primary cause of the inflammation in the cord. Ascending and descending degenerative changes may be noted in the tracts above and below the lesion.

(a) Acute Diffuse Myelitis

Clinical Features.—This form follows exposure to cold, or may be the result of one of the infective fevers. It begins

... soon all four limbs become ... and motor power, and ... the trunk are affected. The ... of urine, involuntary action ... inevitably occurs within one to ... may reach a high degree, and ... the severity of the inflammatory ... marked, and not merely do the ... of the most acute nature ... the sacrum.

~~...~~ The type of myelitis is easy. Its acute- ... being made between *peripheral* ... while in *Landry's paralysis* there

~~...~~ Localised Myelitis

~~...~~ depend on the part of the cord ... the mid-dorsal region. The onset ... and paralysis follows in a ... depends on the extent and position ... is usually transverse, and there- ... of the trunk, or, if the cervical en- ... four limbs may be paralysed as ... sensation. There is a zone of hyper- ... of the lesion, and at the same level ... at all events a feeling of constriction ... the segment or segments of the cord

... the reflexes are at first abolished, and ... except in cases in which, from the ... of the tendon reflexes is per- ... of reflexes is the result of ... the subsequent exaggeration is due to ... the pyramidal tracts. The muscles ... the lesion do not waste, but the muscles ... segments damaged waste and also show the ... In process of time, the muscles ... the lesion tend to become more or less ... of the flexor muscles cause great de- ... convenience to the patient. One frequent ... probably the result of descending degenera-

tion in the pyramidal tracts, is the involuntary twitchings of the affected limbs, which shake the patient, and are very difficult to arrest by treatment. The Babinski sign is often present.

The organic reflexes are seriously interfered with, the patient losing control over bladder and bowels. Retention of urine is often followed by incontinence, and constipation may be associated with absolute incontinence of fæces. Bedsores are extremely common, even although the segments damaged may be few in number, and they may cause the death of the patient from septic absorption.

Should the lesion be situated in the cervical portion of the cord, the arms are paralysed, and in addition vomiting, hiccough, a slow pulse, spinal myosis, dysphagia, dyspnœa, and sometimes syncope may result.

The **Diagnosis** is easy. *Spinal meningitis* causes more marked irritation of the nerve roots, and therefore more pain, but it should be remembered that in myelitis the meninges may be involved as well as the cord.

The **Prognosis** is unfavourable as regards complete recovery, and any evidence of extension of the myelitis upwards is bad. Death is generally due to bedsores, cystitis, or a low type of pneumonia.

Treatment.—Rest of the most absolute description is imperative, and a water-bed should be obtained without delay. The application of ice to the back, the use of mustard applied over the skin, and hot baths are useful methods of treatment, but only under special conditions. A hot bath in an acute case is certainly most beneficial. Ice used with care can do no harm, but counter-irritation such as by mustard or blisters of any kind should never be carried out, without carefully considering the risk of damage to skin whose nutrition may be much below normal. It is well in acute cases to make the patient lie on his face, because by this means the cord is kept as high as possible. The greatest care should be taken of the skin; the parts pressed upon should be washed with soap and water, and then with methylated spirits, and after being carefully dried a dusting powder should be applied, while pressure should be taken off by the use of air-pillows, nests of cotton wool, and similar devices. The bladder should be always remembered, and an aseptic catheter used for drawing off the urine at suitable intervals. Two drugs are of great advant

in many cases, these are potassium iodide and mercury, and with a syphilitic history they may be pushed with more benefit to the patient. Rhus is of advantage where lumbago is occurring. Massage is sometimes useful and also galvanism and mineral to keep up the nutrition of affected muscles. For all the remedies which the patient be directed during the acute stage of the disease. It is hardly necessary to add that the patient should be kept very cool, and that the diet should be composed largely of milk.

Chronic Myelitis

The case being one of acute myelitis in a man of 40 years of age, the following is stated:

"Chronic Myelitis is a disease of the spinal cord, in which the lower part of the cord is affected, and the disease is characterized by a progressive paralysis of the lower extremities, and by a loss of sensation in the lower extremities, and by a loss of reflex action in the lower extremities."

The disease is usually of slow onset, and is characterized by a progressive paralysis of the lower extremities, and by a loss of sensation in the lower extremities, and by a loss of reflex action in the lower extremities."

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called Pott's disease. Pott's disease most commonly affects either the cervical region of the cord or the mid-dorsal. No age is exempt, although the disease usually manifests itself in childhood or youth; probably in not a few cases an injury bears a definite relationship to the commencement of the tubercular process in the vertebral bodies.

The other lesions which may give rise to pressure are tumours, injury, aneurism, and more rarely the presence of hydatid cysts.

Pathological Anatomy.—There is often very marked angling of the spinal canal from collapse of several bodies of contiguous vertebrae, and, in addition, there may be extradural pressure on the spinal cord from tubercular débris. Sometimes meningitis is set up, with or without inflammation of the cord itself.

Clinical Features.—One or two vertebral spines are found to be much more prominent than the others, and tenderness may be felt on pressure. The deformity is gradual in development, and is often associated with pain radiating along the lines of spinal nerves, and due to involvement of the nerve roots. One of the first manifestations of compression of the cord is the development of spasticity in the legs, due to descending degeneration in the pyramidal tracts. This gradually increases until paralysis occurs, but it is a paralysis of the first motor neurone or trophic realm, and therefore unaccompanied by rapid wasting or altered electrical reactions. Generally the Babinski sign is also present. In cases in which myelitis has been lighted up, its phenomena will be superadded, although it is not always easy to recognise the onset.

Where the lesion is in the cervical region of the cord, a retro-pharyngeal abscess is occasionally present, and difficulty may be experienced in moving the head, while sometimes evidence of involvement of the first dorsal nerve may be made out by the narrowing of the palpebral fissure, sinking in of the eyeball, and alteration in size of the pupil.

If the lesion is in the dorsal region of the cord, the curvature is often better marked, and the girdle pains may be very suggestive.

In the lumbar region of the cord the great organic reflex centres controlling bladder and bowels are apt to be involved. A psoas abscess may develop.

Considerable assistance may be afforded not merely by t¹

site of shooting pains, but also by numbness or tingling in areas belonging to affected nerve roots.¹

Diagnosis.—An examination of the back is often sufficient, with percussion of the vertebral spines. The hot-sponge test is frequently of service in recognising a localised myelitis, and care should be taken to examine for the presence of tumours of breast or other region, or of tubercular manifestations in the chest or elsewhere.

Prognosis and Treatment.—The treatment of tubercular caries of the spine is essentially surgical. There are many cases in which, after a prolonged period of rest in bed, often with the application of splints and other devices to remove pressure from the affected vertebrae, a process of cure results, which, if it does not imply the complete elimination of the tubercle bacillus, at all events permits of the patient leading a useful and often prolonged life with little physical discomfort. It is important, in cases of cervical caries, to prevent the rotation of the head, and therefore sandbags have been used with considerable effect during the period of rest in bed, or a suspensory apparatus by means of which the weight of the head is supported, and rotatory movements largely prevented. Poroplastic jackets are often worn in vertebral caries occurring at a lower level.

In very early cases operative treatment is not necessary, and rest is alone sufficient; but in cases in which there is marked angular curvature with, in addition, pressure of tuberculous débris on the outside of the dura mater, operation by laminectomy may alone yield any hope of cure. The spastic paralysis may gradually disappear, and recovery follow. The ordinary open-air, good food, and tonic treatment for tubercular subjects should be carried out in cases of tuberculosis of the vertebrae.

(12) HÆMORRHAGE INTO THE SPINAL MEMBRANES (HÆMATORRHACHIS). HÆMORRHAGE INTO THE SPINAL CORD (HÆMATOMYELIA)

I. HÆMORRHAGE INTO THE SPINAL MEMBRANES.—This may occur either outside the dura or inside between the membranes

¹ Pressure on nerve roots may cause intensely painful areas of skin, which are nevertheless anæsthetic to tactile sensibility and common sensation. This is called *anæsthesia dolorosa*.

and the spinal cord. *Extra-meningeal hæmorrhage* may be the result of injury, and the hæmorrhage may press severely upon the dura and the spinal cord within. An aneurism of the third part of the arch or of the descending aorta has been known to erode the vertebral bodies and to rupture into the spinal canal, causing death by the great pressure which it produces.

Intra-meningeal hæmorrhage is much more frequent, and may be due to blood passing down from the base of the brain. Such hæmorrhage may originate in the ventricles from rupture of an ordinary intracerebral hæmorrhage into the neighbouring lateral ventricle, or an aneurism, sometimes involving the circle of Willis, may rupture; while from fracture or other injury, severe hæmorrhage, either subarachnoid or subdural, may be caused. In all of these cases the blood may flow downwards into the spinal canal. In death from tetanus, strychnine poisoning, or from a severe convulsive seizure, submeningeal hæmorrhage may occur. It is common to find in cases which may be classified as toxic, and especially in the infective fevers of malignant type, that numerous small hæmorrhages are present, sometimes under the pia and sometimes subarachnoid in position, and these may be found in connection with both brain and cord.

The **Clinical Features** dependent on the meningeal hæmorrhage vary considerably in different cases. There may be sharp and severe pain, with evidence of irritation of spinal nerve roots; in other cases the pressure is sufficient to induce paralysis, and the organic reflexes may be completely abolished. There is sometimes muscular spasm, sometimes complete paralysis, and in not a few cases there may be absolute coma.

A special form of internal pachymeningitis associated with hæmorrhages not infrequently affects the cervical meninges. It is described under meningitis (p. 596).

II. HÆMORRHAGE INTO THE SPINAL CORD.—This is a rare condition, although more common in males, and appears to be most frequently due to injury, such as a bullet wound or a severe blow, or even forcible bending of the neck. It has been described in women as occurring in labour, and in both sexes it may be associated with alcoholism, tetanus, strychnine poisoning, and severe convulsions. It seems likely that in a number of cases the hæmorrhage is in reality secondary to, and occurring in, a glioma of the cord, and it is hardly necessary to add that in myelitis small hæmorrhages are common.

Pathological Anatomy. — The hæmorrhage varies in position and in extent; it may rupture through the cord, it may tear it up transversely, or it may burrow vertically, and especially is this vertical burrowing apt to occur in connection with gliomatous tumours.

The **Clinical Features** vary considerably. The onset is much more sudden than in meningeal hæmorrhage, and the paralysis, sensory and motor, may involve the legs alone, or the arms and legs, according to the position of the lesion. If the hæmorrhage is primary, a myelitis almost invariably follows, and death, if not due to the hæmorrhage itself, is generally not long delayed. In slight cases the blood may be absorbed, and partial recovery results.

The **Diagnosis** is usually easily made by the suddenness of onset and the rapid trophic changes which generally ensue. Lumbar puncture is of the greatest value in enabling the physician to diagnose the presence, not merely of a little blood in the cerebro-spinal fluid, but of an extravasation almost entirely hæmorrhagic.

The **Prognosis** varies greatly. In hæmorrhage into the membranes relief may sometimes be obtained by operation. Hæmorrhage into the cord is usually early fatal.

Treatment. — The patient should be placed in the prone position, and if possible on his face. Ice should be applied along the line of the spines, and ergotine administered. Once the hæmorrhage has ceased, it may be prudent to canvass the possibility of operation.

(13) TUMOURS OF THE SPINAL CORD AND MEMBRANES

Etiology. — With the exception of syphilitic and tubercular cases the origin is doubtful: possibly injury may be an important factor in some instances.

Tumours of the Cord. — These are rare: they include gliomata, sarcomata, tubercular nodules, gummata, and hydatid cysts.

Tumours of the Membranes. — These include gummata, sarcomata, myxomata, tubercular nodules, hydatid cysts, fibromata, psammomata, and lipomata.

Emacars and tubercular disease of the vertebral bodies and aneurismal tumours may also cause pressure on the cord.

The **Clinical Features** vary greatly, depending much on the extent of the cord involved, the amount of pressure on the nerve roots, and the degree of meningitis present in each case. If the tumour is in the cord, it may interfere with grey and white matter, and may induce all kinds of changes, possibly a hemi-lesion, or more likely a complete transverse lesion. If the tumour is situated in the membranes, or grows from the vertebral bodies, there is apt to be pressure on the nerve roots, with pain along the line of the affected spinal nerves. There is generally a localised myelitis in the neighbourhood of the tumour, and in cases of glioma, hæmorrhage often occurs into the tumour tissue, greatly shortening the patient's life. Sensory and motor symptoms will depend on the site, extent, and nature of the tumour.

Diagnosis.—A careful examination should invariably be made of the vertebral spines, and by tapping with a percussion hammer, pain may be elicited over one or more vertebræ, indicating the position either of the lesion or of the myelitis to which it has given rise. A history of syphilis and evidences of tubercle in other parts of the body may be helpful, but often the diagnosis is difficult.

The **Prognosis** depends largely on the nature of the tumour; in certain cases surgical interference is possible. In other cases, and especially where the lesion is syphilitic, potassium iodide and mercury may be administered, while in tubercular disease of the vertebral bodies prolonged rest in the prone position and sometimes surgical treatment are beneficial. Tumours of the cord itself are of necessity very grave.

The **Treatment** has been indicated. In tubercular and syphilitic cases much may be done in the way of attempted cure, and in tumours of the membranes surgery offers some hope of relief, but tumours of the cord are, as a rule, absolutely incurable. The bladder must be watched, and where necessary the urine drawn off. Bedsores should be carefully obviated, and, where they develop, treated with antiseptic precautions. Sometimes the patient requires the administration of morphia to relieve pain.

(14) SCLEROSIS FROM TOXIC BLOOD POISONING

In pernicious and other profound anæmias, and sometimes in diabetes, sclerosis may occur.

Pathological Anatomy.—It is an increase of neuroglia replacing degenerated nerve fibres, and involving the anterior, lateral, and posterior columns, and specially marked in the cervical region of the cord. The affection diminishes in intensity from above downwards, and the lumbar region appears to escape to a great extent. There is no special change in the cells of the grey matter, but there is almost certainly a parenchymatous degeneration of the nerve fibres. Endogenous tracts such as the septo-marginal do not suffer.

Clinical Features.—After a preceding history of weakness and ill-health the patient begins to suffer from locomotor-like pains and loss of the knee-jerks. In other cases there is spasticity, and the arms may be involved as well as the legs. Choreic movements are not uncommon, and in severe cases bedsores and cystitis develop.

Diagnosis.—The disease resembles *locomotor ataxia*, *spastic paralysis*, and *peripheral neuritis*, but the Argyll Robertson pupil is absent, and the investigation of the blood in cases of pernicious anæmia yields distinctive results.

The **Prognosis** is unfavourable, and the **Treatment** must be on general lines, iron and arsenic being of most benefit, while rest in bed should be enjoined.

(15) CAISSON DISEASE. DIVER'S PARALYSIS

A disease due to high atmospheric pressure, of at least 1, generally 2 to 4 atmospheres.

Etiology.—The *predisposing* causes are, any lowered vitality, especially alcoholism, obesity, and diseases of the heart and kidney. Inexperienced workmen suffer most, but even those who are experienced cannot remain in a caisson at a great depth for more than a very limited time.

The phenomena are thought to be due to escape of gas, possibly nitrogen, from the blood when the pressure is taken off.

Pathological Anatomy.—Hæmorrhage and congestion have been described in acute cases, and certainly myelitis in cases fatal after some time, but the gross lesions are very indefinite. Leyden has described spaces in the cord.

Clinical Features.—At the onset there may be pains in the joints and muscles. The more serious symptoms are

cerebral and *spinal*. The *cerebral* include faintness, with nausea and vomiting, headache and giddiness, sometimes coma, and occasionally apoplectiform seizures, which may be instantly fatal. The *spinal* include neuralgic pains, often very severe and often in paroxysms, felt in the legs, arms, back, etc., increasing weakness, and later paralysis.

In the majority of cases only certain of these symptoms appear, and they may do so immediately, or some time after the patient has left the compressed atmosphere.

Prognosis.—In most cases in recovery; where the more severe phenomena appear, paralysis or death.

Treatment.—No one should enter the caisson who feels seedy, and all new-comers should work for a very short time at first, and never at a great depth. The greater the pressure the shorter should the time of work be. For the intense pains give opium, and treat on general principles.

(16) SYRINGOMYELIA

The formation of a cavity or cavities in the spinal cord, associated with the presence of more or less gliomatous embryonic tissue, in which the cavity formation may originate.

Etiology.—It is probably a congenital condition dependent on the persistence of embryonic tissue, but it may also be due to hæmorrhage into, or possibly injury of, the spinal cord. Perhaps, also, in rare cases there may be persistence of the primitive tube, but this seems a less likely explanation than the presence of embryonic gliomatous tissue.

Pathological Anatomy.—The cavity or cavities may be connected with the central canal, or may be distinct. They may extend up into the medulla and pons, and in the cord are most commonly met with in the cervical enlargement or the upper dorsal region. Gliomatous tissue surrounds the cavity in varying amount, and the cavity usually enlarges, and when it does so, causes pressure on neighbouring structures. The simplest form of cavity is a dilatation of the existing canal of the cord, and is probably dependent on an embryonic error of development, although possibly also on an excess of cerebro-spinal fluid. In many normal cords in the adult the central canal becomes obliterated without any morbid condition to account for such obliteration, but it is incorrect to ascribe

that the mere persistence of the canal constitutes a cavity such as we find in syringomyelia. The embryonic tissue surrounding the cavity or cavities is translucent in appearance and retains much of its embryonic character, and sometimes this embryonic tissue is found at a lower level than that to which the cavity may extend. The posterior cornu is not infrequently involved by cavity formation.

Both grey and white matter are liable to pressure when the cavity enlarges, and it is to this pressure that the typical clinical features are due.

Clinical Features.—Between the 20th and 30th year the symptoms and signs of syringomyelia generally develop, and probably therefore at the time when the strain of life becomes greatest. Its onset is insidious, and the following features are present:—

(1) Loss or diminution of temperature sense and common sensation, sometimes involving both arms, often more marked in one. In other cases areas of cutaneous anæsthesia develop and gradually extend.

Tactile and muscular sense impressions are usually unaffected.

(2) Atrophy of certain muscles frequently occurs, beginning in the small muscles of the hand and extending upwards, sometimes involving special groups of muscles, and dependent on the amount of interference with the anterior horn.

(3) Spastic phenomena develop in the legs, the result of pressure on the pyramidal tracts, with exaggerated tendon reflexes and ankle clonus, and the Babinski sign is positive.

(4) Marked trophic changes occur in certain bones and joints of the affected limb or limbs. The bones become brittle, and the joint change may resemble what is sometimes met with in locomotor ataxia.

(5) In certain cases of syringomyelia, trophic changes also occur in the skin of the hands and in the nails. The skin becomes glossy, the nails thickened, brittle, and with a tendency to drop off. Deep-seated ulcers or painless whitlows sometimes develop, and eczema, herpes, and other skin affections have been noted, while localised sweating is occasionally present.

The electrical examination may show the reaction of degeneration in wasted muscles; fibrillary twitchings and tremors may be present, similar to what is seen in progressive muscular

atrophy. The spinal muscles may be weakened and scoliosis results.

If the lesion is situated in the lumbar enlargement, the organic reflexes may be abolished, and bedsores, cystitis, and incontinence of the bowels occur. Where a cavity extends into the medulla and pons, bulbar phenomena are apt to appear, such as difficulty of swallowing and of respiration, and enfeeblement of heart's action.

Diagnosis.—*Leprosy*, although it may be associated with painless ulcers, is absolutely distinct from this disease. A localised *myelitis* or a *cervical pachymeningitis* may each cause symptoms so far resembling syringomyelia, but in cervical meningitis the retraction of the head is suggestive. It is possible that in *gliomatous tumours* of the cord cavities may arise, and so phenomena resembling syringomyelia may develop, but the slowly progressive clinical features of a typical syringomyelia are very different from the rapid advance of a gliomatous tumour.

Prognosis.—Sometimes the disease becomes stationary, not merely for months but for many years, and in such cases there may be a moderate improvement, and the trophic affections are more likely to be amenable to treatment.

The **Treatment** must be on general lines. No surgical procedure can effect a cure, and as the disease is embryonic in origin, drugs are of little avail. Attention should be paid to the treatment of trophic sores and skin affections.

MORVAN'S DISEASE.—This is really included under syringomyelia. It is an affection in which, along with neuralgic pains, there is wasting and paralysis of muscles of the hand and forearm, with anæsthesia and analgesia, and the formation of small cold abscesses over the distal phalangeal joints. Sloughing may result from these anæsthetic whitlows, and necrosis of bone is not infrequent, causing serious deformity. The disease is closely allied in appearance to the anæsthetic type of *leprosy*, but in that affection the pigmentation of the skin and the course of the disease are typical and cannot well be confused.

Morvan's disease is due to cavities in the cord similar to those described in syringomyelia.

(17) SPINA BIFIDA

It is important to consider spina bifida in its relationship to syringomyelia. It is defective closure of the bony canal containing the spinal cord. The membranes lining the cord bulge into the sac, which is distended with cerebro-spinal fluid. It is usually found in the lumbar region of the spine, and in a considerable proportion of cases the central canal of the cord is enlarged in connection with the presence of the meningocele. This enlargement of the central canal is common above and sometimes below the site of the protrusion of the sac, but generally the central canal of the cord does not communicate with the sac, although in a few cases it does.

Clinical Features.—The symptoms depend on the interference, if any, with the spinal cord. These include paralysis, possibly atrophy, and perhaps sensory changes, as a rule involving the legs only. Talipes varus is the most frequent result of such muscular change, the anterior tibial muscles being generally affected.

The **Prognosis** depends on the possibility of surgical treatment.

The **Treatment** is purely surgical, and usually the sac is tapped. The cases in which the cord is also affected are eminently unsatisfactory.

V. DISEASES OF THE MEDULLA AND PONS

GLOSSO-LABIO-LARYNGEAL PARALYSIS (BULBAR PARALYSIS)

THE term bulbar paralysis implies a lesion of nerve centres in the medulla, although nuclei in the pons may also be involved. The result of the lesion is paralysis, more or less complete, of the muscles governing speech, including phonation, and of those which have to do with the acts of swallowing and mastication; while more rarely the eye muscles and other muscles innervated by cranial nerves become affected (see Diagram 31, p. 563).

The lesions in question include hæmorrhages, acute inflammations, or patches of sclerosis involving the nerve nuclei,

although perhaps the most important, because the most common, group of cases depends in reality upon the action of a toxin similar to that which causes progressive muscular atrophy. The action of this toxin is probably limited to the nerve cells. Such lesions may be secondary to progressive muscular atrophy, or may develop primarily in the medulla and pons.

I. ACUTE BULBAR PARALYSIS

This appears to be sometimes the result of influenza or some other continued fever; it may be hæmorrhagic in nature, or it may be a polio-encephalitis akin to polio-myelitis in the cord. Either the hæmorrhage or the encephalitis may involve the higher nuclei, as, for example, those of the 3rd and 4th nerves, or may be limited to those in the medulla and pons; even in the cases where the lesion is probably an encephalitis, minute hæmorrhages are extremely common.

The onset is acute and the muscles may be extensively involved; where the higher nuclei suffer, certain of the eye muscles are paralysed, while not infrequently there is nystagmus and a cerebellar gait. Where the lesion is in the pons or medulla, there is more or less paralysis, often of all four limbs, sometimes a hemiplegia and sometimes a crossed paralysis. The latter condition is dependent on a hemi-lesion in the lower half of the pons, by which the motor fibres to the face are affected on the side of the lesion, while the fibres to the opposite arm and leg are also affected (see Diagram 44 on page 646). Extensive lesions of such an acute nature in the medulla or pons are incompatible with prolonged life, and the disease soon terminates fatally.

There are instances in which acute softening is the result of embolus or of syphilitic endarteritis in connection with some small nutrient artery supplying that region. In these cases the paralytic phenomena depend on the position of the patch of softening. There is generally marked headache, giddiness, and loss of consciousness, while vomiting and sometimes convulsions may occur. It is always possible to have paralysis of the respiratory and cardiac centres, which may suffer in the same way as the nuclei or fibres of the cranial nerves. Hæmorrhages are by no means uncommon in connection with patches of softening, and either an extensive

hæmorrhage or a progressive thrombosis may rapidly terminate the patient's life. Many of these cases are of syphilitic origin, and where treatment is possible, they should be treated by the usual antisyphilitic remedies.

II. CHRONIC BULBAR PARALYSIS

Etiology.—A disease of middle life, more frequent in men, and generally in those of neurotic family history; possibly dependent on mental over-strain, exposure, syphilis, and lead poisoning, while it may develop as an extension upwards of the lesion of progressive muscular atrophy.

Pathological Anatomy.—There are marked degenerative changes in the nuclei of certain nerves in the medulla, and especially the hypoglossal, the facial, the glosso-pharyngeal, the spinal accessory, and the vagus. The nerve-endings in the affected muscles degenerate, and so do the nerves and the muscle fibres to which they are distributed. The nerve-cell changes in the nuclei are typical, the cells atrophy and certain of them disappear, while in others pigmentary changes have been described, and there is disappearance of the chromatic substance from the cell protoplasm and diminution in the number of grey processes. The affected nerve-roots are grey and shrivelled, and many nerve fibres in them are found to be degenerated.

Clinical Features.—The development is insidious, and begins with indistinctness of speech from weakness of the tongue and lips: the letters l, n, r, and t are among the first to be affected, while later on o, u, p, b, and m are pronounced with difficulty. The palate becomes weak, fluids tend to regurgitate through the nostrils, and the patient speaks with a nasal tone. Soon afterwards swallowing becomes difficult, partly because the tongue is unable to perform its usual function in moving the bolus of food backwards towards the œsophagus, but also because the œsophageal muscles themselves are weakened. The glottis may not close, and in time food tends to enter the larynx, either causing fits of coughing by which the foreign body is expelled or else setting up a septic pneumonia if inspired. Paralysis of the larynx indicates the involvement of the nuclear cells governing the laryngeal muscles, and the patient becomes apnoeic. When

this stage is reached the tongue is usually completely paralysed, cannot be protruded from the mouth, and becomes wrinkled, and fibrillary tremors may be seen in it. The lower lip hangs down, and thick, tenacious saliva tends to dribble more or less constantly. The electrical excitability of the affected muscles is diminished, and there may be a partial reaction of degeneration. The patient's face hardly demonstrates the fact that in mind and intelligence he is as acute as ever, and often keenly feels his disability as regards speech and deglutition. There is no change in sensation, and, as a rule, the organic reflexes are unaffected.

The disease is a progressive one, and serious involvement of the respiratory and cardiac centres in the medulla may lead to a fatal result, or septic pneumonia already mentioned may terminate the patient's life.

Diagnosis.—The lesion is one of the second trophic realm. It is localised, and usually the nuclei become involved in the order mentioned in describing the clinical features. In a considerable proportion of cases the disease has commenced with progressive muscular atrophy, and only in the later stages do the bulbar phenomena develop.

The **Prognosis** is bad, and as a general rule nothing can be done to prolong life.

Treatment.—Benefit might be hoped for should the lesion be syphilitic in origin. Keep the patient at rest and avoid excitement of all kinds. The feeding is most important, especially in cases where there is the slightest risk of food entering the larynx. Electrical treatment should be tried, and especially galvanism, while strychnine, arsenic, phosphorus and silver have proved beneficial in a very few cases.

VI. DISEASES OF THE BRAIN

Introductory.—A series of diagrams shows the convolutions of the cerebrum, the blood supply of these convolutions, and the very important relationship of the blood supply to the great motor and sensory areas of the cortex. A description is given under regional diagnosis of the results of cerebral lesions, with the position of the chief centres, while the sensory and motor centres governing the reception of symbols and the

evolution of speech are discussed under aphasia. A diagram of the circle of Willis with the nutrient arteries which come

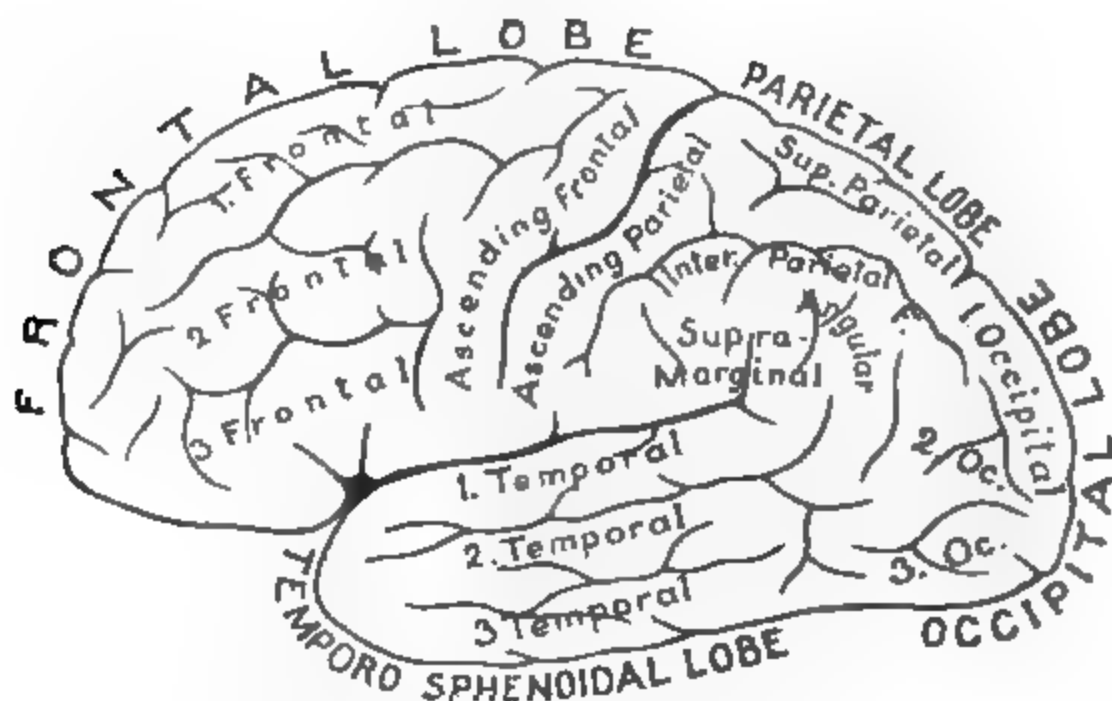


DIAGRAM 38.—Diagram showing Cerebral Convolution.

off from it for the supply of the region of the internal capsule is shown on p. 654.

1. REGIONAL OR TOPICAL DIAGNOSIS OF CEREBRAL LESIONS

The Cerebral Cortex

(1) **FRONTAL LOBE.**—The pre-frontal region has been termed a silent area, while the post-frontal region is associated

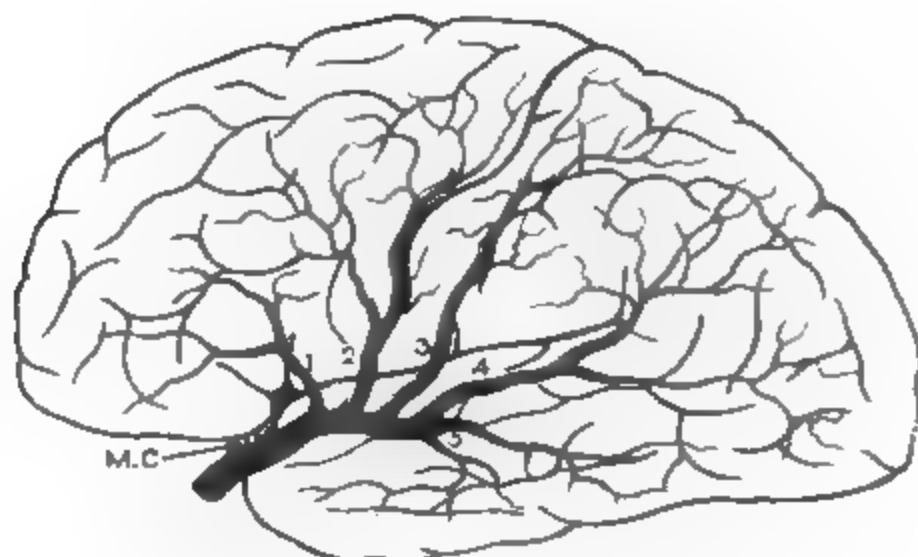


DIAGRAM 39.—Cerebrum showing distribution of Middle Cerebral Artery.

with conjugate deviation of the eyes. Irritation of this centre causes deviation of the eyes to the opposite side from the

lesion, whereas a destructive lesion causes conjugate deviation towards the side of the lesion.

The frontal lobe appears to be associated in some way with the intellect and memory, because disease of the frontal region causes failure of memory, hebetude, sleepiness, and in cases in

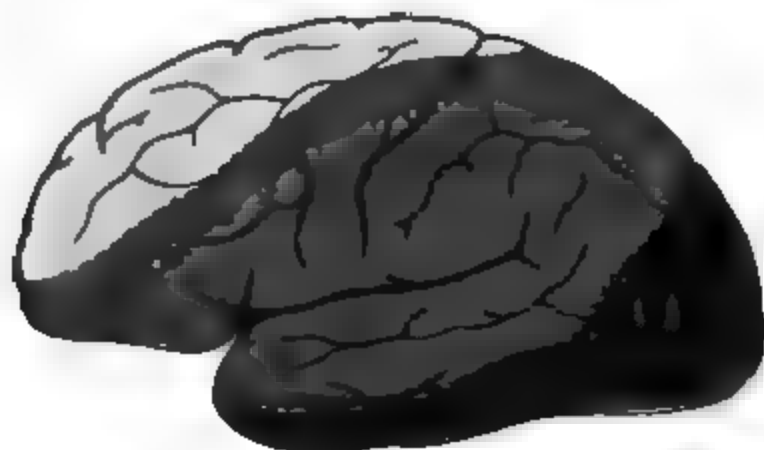


DIAGRAM 40.—Cerebrum, showing area of blood-supply from Anterior, Middle, and Posterior Cerebral Arteries.

which the affection is less marked a great inability to concentrate the mental faculties. It must, however, be admitted that in certain cases extensive lesions of the frontal lobe have been present without any definite mental feebleness.

The following are the common lesions met with in the

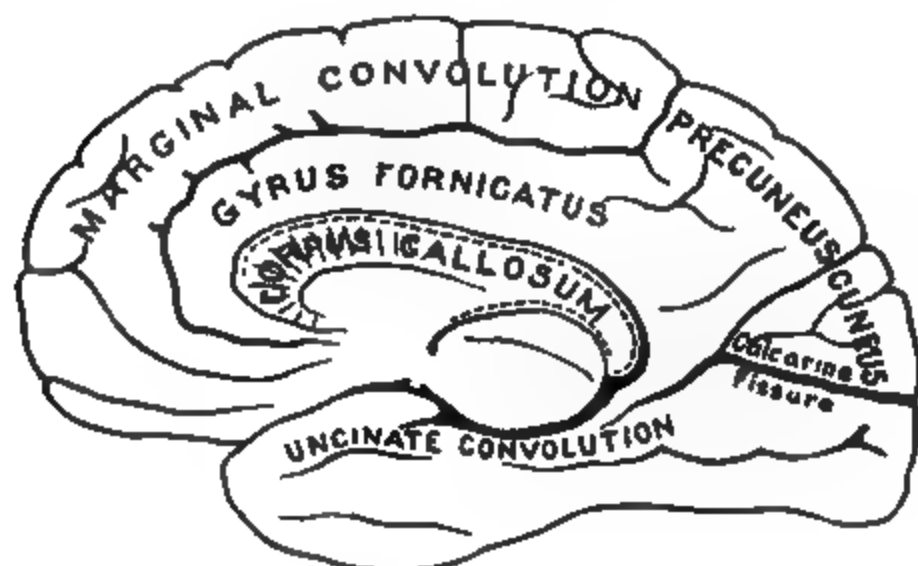


DIAGRAM 41.—Internal surface of Cerebrum.

frontal lobe—tumour, abscess, and hæmorrhage from the anterior cerebral artery; but the last-named is rare, and is more commonly the result of trauma.

(2) THE ROLANDIC REGION—(a) *An Irritative Lesion.*—This causes epileptiform convulsions on the opposite side of the body from the lesion, and it may be limited to one arm, one

leg, one group of muscles, etc. (monospasm); in other cases the spasms become general, and may be associated with loss of consciousness. There may be no organic lesion, unless some paralysis develops, but in most cases there is a subsequent and temporary motor paralysis, and sometimes tingling or numbness of the affected limb or part of a limb. The group of clinical features we have just described constitutes Jacksonian epilepsy, and the commonest causes are tumour, a syphilitic lesion such as a gumma, occasionally patches of softening, and more rarely hæmorrhage.

(b) *A Destructive Lesion.*—This may be the sequel to an irritative lesion; a tumour or hæmorrhage may first cause irritation and later paralysis. The phenomena are paralytic

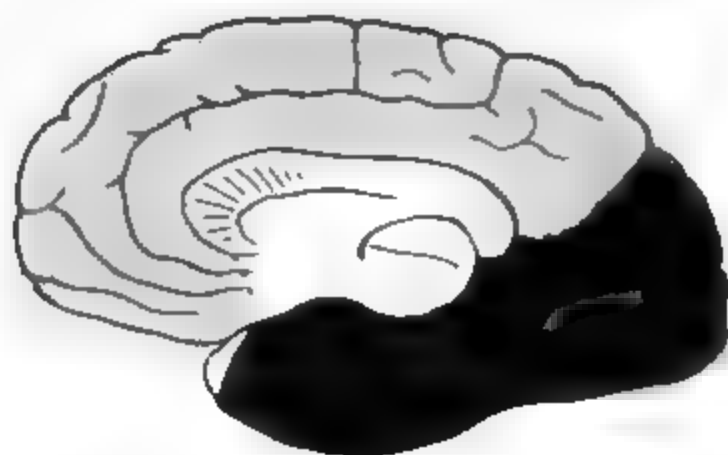


DIAGRAM 42.—Internal surface of Cerebrum showing supply of Anterior Cerebral in yellow, Posterior Cerebral in blue, and Middle Cerebral uncoloured.

and involve the muscles on the opposite side of the body from the cortical centre or centres which have been destroyed. This is a lesion of the first or upper motor neurone or first trophic realm, and therefore there is no electrical change and no rapid muscular wasting. The lesion, if extensive, closely corresponds to a lesion in the internal capsule, but in the region of the cortex a large area requires to be involved in order to produce a complete hemiplegia. There may be therefore a succession of attacks, with a gradual involvement of more and more of the cortical centres: paralysis of one limb alone (monoplegia) generally suggests that the lesion is cortical. Where the cortical lesion is on the left side of the brain and involves Broca's convolution, motor aphasia results if the individual is right-handed.

It has lately been demonstrated, at least in monkeys, that the motor areas lie in front of the fissure of Rolando, and that

the ascending parietal convolution and the superior parietal lobule are really sensory cortical centres as represented in Diagram 43.

(3) PARIETAL LOBE.—The superior parietal region was believed to be associated with the ability to recognise the shapes of things, or what is termed the stereognostic sense, but probably this is inaccurate. A lesion of the inferior parietal lobule may yield no definite sign, or there may be some ptosis, hemianopsia, and, if it is on the left side of the brain, also word-blindness. The relationship of ptosis to the occipital gyrus lacks confirmative evidence.

(4) OCCIPITAL LOBE.—A lesion of the occipital lobe causes

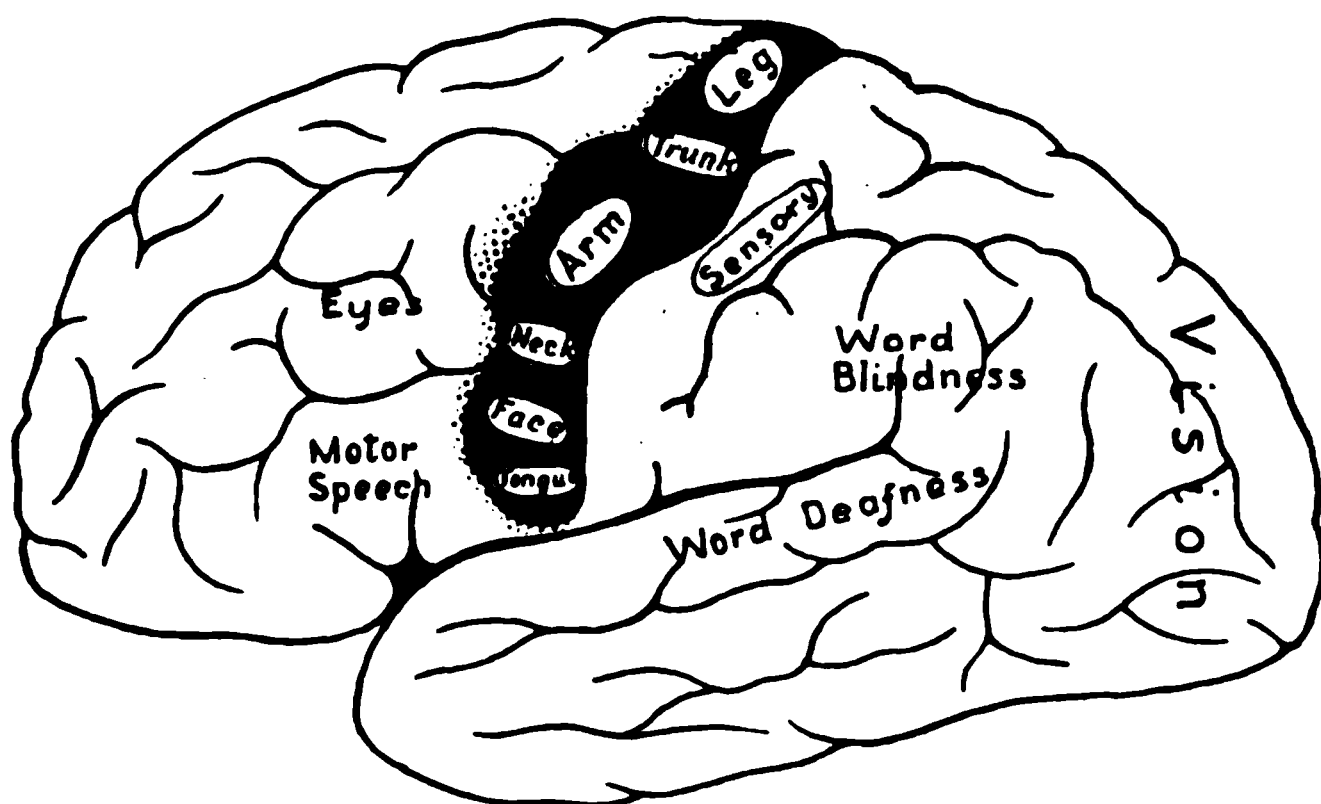


DIAGRAM 48.—Left-half of Brain showing Cortical Motor (in red) and Cortical Sensory (in yellow) areas. It also shows the position of the visual, auditory, and speech centres.

homonymous hemianopsia which may be associated with a hemiplegia and also a hemianæsthesia, provided the internal capsule is also involved; if the lesion extends to the left angular gyrus, then word-blindness is associated with the hemianopsia.

(5) TEMPORAL LOBE.—There may be nothing, or deafness, if the middle and posterior portions of the superior temporo-sphenoidal convolution are involved, while if the lesion is on the left side of the brain there is word-deafness. The cortical centre for the sense of smell is believed to be located in the uncinate convolution.

The Hippocampal Gyrus (posteriorly) and the Gyrus Fornicatus.—These were considered until recently to be the centres

for tactile and general sensation, and possibly also the centres for recognition of the position of the limbs and muscles, but the parietal region is probably the chief sensory centre.

The Corpus Callosum.—The commissural fibres joining opposite cortical centres pass through this structure. Bristowe has described four cases of tumour in which the lesion was found to be in the corpus callosum. In all these cases they were (1) ingravescent; (2) gradual in onset, with hemiplegia, better marked on the one side, but also vaguely present on the other; (3) they showed drowsiness, apathy, difficulty in swallowing, and speechlessness; (4) no cranial nerves were involved; (5) death occurred from coma. There was no sickness, no headache, and in one case out of the four no optic neuritis.

Centrum Ovale and Internal Capsule.—We cannot distinguish destructive lesions of the centrum ovale from those of the Rolandic cortex, but irritative lesions show certain differences. If they are cortical, there are tonic followed by clonic convulsions. If, on the other hand, the lesion is situated in the centrum ovale, there are only tonic and no clonic movements, probably because the stimulus must be continuous in order to get clonic spasms. If a hæmorrhage is immediately below the cortex, we may have a typical history of cerebral hæmorrhage with, in addition, an element of spasm. Convulsions and early rigidity in cerebral hæmorrhage may be due to pressure on the fronto-parietal fibres, and not to hæmorrhage into the lateral ventricles.

The position of the motor fibres in the anterior two-thirds of the posterior half of the internal capsule, and of the sensory fibres which are supposed to be in the posterior one-third, as well as the relationship of the optic radiation of Gratiolet, may be seen in Diagram 46, and reference will be made to the usual site of a cerebral hæmorrhage in connection with that affection.

Corpus Striatum, or the Basal Ganglia.—*The caudate nucleus*, according to Ferrier, governs in some way the movements on the opposite side of the body; but softening or tumour has been found in both the *caudate* and *lenticular* nuclei, and yet no paralysis resulted. A hæmorrhage into the head of the caudate nucleus causes hyperpyrexia, and the hæmorrhage is most generally from one of the antero-median nutrient arteries (see Diagram 47).

Optic Thalamus.—We do not know its function, but hemianæsthesia is often associated with a lesion involving it, and not infrequently the optic radiation of Gratiolet is also affected, causing hemianopsia. From the fact that lesions in or near the optic thalamus are found to be associated with athetosis, it seems probable that mimetic movements of the face and possibly of the limbs depend on cortical fibres passing to the optic thalamus.

The Crura Cerebri.—Lesions limited to the crura cerebri or to one crus are rare. Tumours, generally tubercular in nature, or gliomata, may invade the crura cerebri, and neoplasms growing from the base of the skull have also sometimes involved this structure. Hæmorrhages, thrombotic softenings, and abscesses are rare. The sensory fibres pass in the tegmentum, and the motor fibres in the middle portion of each crus. A form of alternate or crossed paralysis, consisting in the arm and leg and often the lower facial region being paralysed on the opposite side from the lesion and the 3rd nerve on the same side, may be produced by a unilateral involvement of the crura cerebri. The lesion of the 3rd nerve is not always complete.

Tumours involving the crura cerebri readily involve both 3rd nerves, and may also affect both motor tracts. A lesion of the tegmentum causes anæsthesia.

The Corpora Quadrigemina.—These structures are too small in extent to be alone involved by hæmorrhages, tumours, or softening, and therefore it is only by a study of different cases that we can arrive at the correct phenomena to which their involvement gives rise. There is an unsteady, reeling gait much like the drunken gait in a case of cerebellar tumour. There is often also ophthalmoplegia, generally incomplete, and due to an affection of the subjacent 3rd nuclei. Irritation of the corpora quadrigemina on one side causes dilatation of both pupils, and especially that of the opposite eye; and it must be remembered that there are important connections between the fibres subserving vision and these bodies (p. 564).

The central tracts of the cochlear nerve ascend through the opposite lateral fillet, the posterior tubercle of the corpora quadrigemina, the brachium and the internal geniculate body, and thence to the internal capsule. Therefore hearing should be tested in all cases of supposed lesion of the corpora quadrigemina, and, generally speaking, it will be involved

side opposite to the tubercle affected (in 19 cases of tumour of the corpora quadrigemina, 9 showed a definite defect of hearing).

In tumour cases other structures are invariably involved, such as the optic thalamus, the tegmentum, pons, or the superior cerebellar peduncles. In experimental cases tetanic spasms are caused by irritation of the corpora quadrigemina, and the knee-jerks have been found to be either normal or more generally exaggerated; in certain cases they are abolished.

The Pons.—The symptoms vary according to the position and extent of the lesion. Tumours are occasionally met with, but gliomata may infiltrate the whole pons, and hæmorrhages, unless small, rapidly prove fatal. A lesion of the tegmentum tends to involve the 5th, 6th, 7th, and 8th nerves, and also sensation and co-ordination of movements, whereas a crustal lesion causes motor paralysis of the limbs, and involves the 7th, 10th, 11th, and 12th nerves.

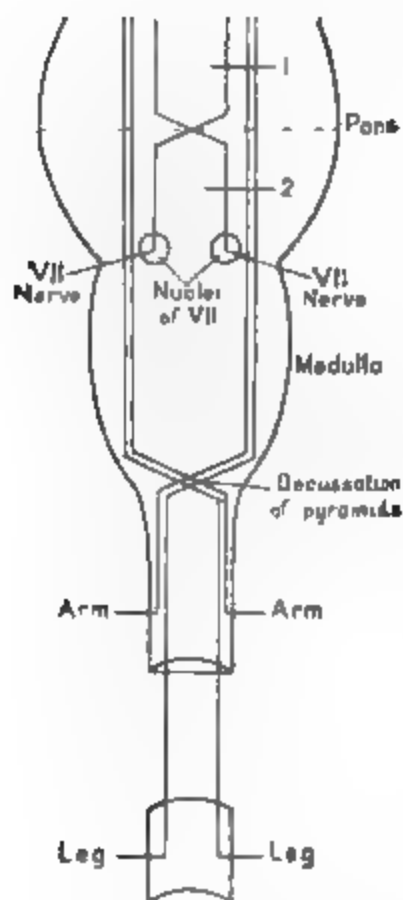


DIAGRAM 44.—Schematic Diagram showing Decussation of Face (7th) Fibres in the middle of the Pons and the Arm and Leg Fibres at the Pyramidal Decussation in the Medulla. Lesion 1 would give paralysis of face, arm, and leg on the opposite side from the lesion. Lesion 2 would give Crossed Paralysis, the face being paralysed on the same side as the lesion and the arm and leg on the opposite side.

The chief clinical features of a pontine lesion are profound coma, and complete relaxation of the muscular system, with, however, a tendency to tonic or clonic spasms, sometimes irregular and unilateral. There is often hyperpyrexia, the temperature even reaching 109° Fahrenheit, but only in cases where the lesion is sudden as in hæmorrhage. Pin-point pupils are almost invariably present.

Crossed paralysis depends on the level of a lesion which is pontine and unilateral. The fibres from the face centre decussate in the middle of the pons on their way to the 7th nucleus, and therefore a lesion above the middle of the pons will give paralysis of the face, arm, and leg on the opposite side from the lesion, whereas

a lesion in the lower half of the pons will give par

arm and leg on the opposite side from, and of the face fibres on the same side as, the lesion. Similarly the 5th and 6th nerves may be affected, as well as the 7th on the same side as the lesion, and the limbs and tongue on the opposite side. The involvement of the tongue depends on whether the lesion extends to the fibres or nucleus of the 12th nerve in the medulla. Hearing is not infrequently affected on one or both sides, and from the small size of the pons and the close relationship of motor and sensory tracts hemianæsthesia or bilateral anæsthesia may be present. There is often hemianæsthesia of the body on the opposite side from, and of the face on the same side as, the lesion, depending on whether the lesion is above or below the junction of the ascending and descending roots of the 5th nerve. There are sometimes ataxic disorders associated with pontine lesions, but the position of the fibres to which the phenomena are due is somewhat doubtful. Articulation and deglutition may be affected; interference with articulation probably depends on the involvement of the central fibres of the 12th nerve above the nucleus.

Cerebellum.—The common lesions in the cerebellum are tumours, abscess (secondary to middle-ear disease), and in young persons hæmorrhage. The clinical features of cerebellar disease may be latent, but in most cases there are the following very definite and suggestive phenomena. Instability when attempting to stand or walk is peculiarly typical. The gait resembles that of a drunk man, although curiously enough when the patient lies down the finest movements may be possible without any inco-ordination. It seems probable that the middle lobe of the cerebellum is the special centre for co-ordination. We sometimes find also rotatory movements believed to be associated with the cerebellar peduncles, but these movements are not constant and are of doubtful significance. The cerebellum governs combined and co-ordinated muscular movement, and for this the cerebrum and cerebellum are both needed and the integrity of the tracts joining these important centres with each other. Paralysis may occur in cerebellar lesions, but there is no special site, and it is probably the result of pressure. Convulsions are not uncommon on the side of the lesion. The knee-jerks vary; they may be increased, diminished, or sometimes normal. There is occasionally tonic contraction of the neck muscles, retraction of the head, flexion

of the arms, and extension of the legs, probably the result of indirect irritation. The pupils may be pin-point or dilated, and there is often the Argyll Robertson phenomenon. Nystagmus is extremely common, and optic neuritis is practically constant in the case of cerebellar tumours. Hearing, smell, taste, and cutaneous sensation are generally unaffected; the pulse is sometimes quickened, sometimes slowed; but the significance of this last phenomenon is unknown.

2. APHASIA

The following varieties of aphasia occur:—

(1) *Motor Aphasia*.—(a) Motor Aphasia proper, or difficulty in expressing by words spoken (symbols) ideas or facts. The motor speech centre is in the posterior third of the inferior frontal convolution usually called Broca's convolution, and in right-handed individuals it is situated on the left side of the brain.

(b) Agraphia or an affection of the writing centre, which is certainly situated in the cortical area governing movements of the hand, implies a greater interference than that merely affecting mechanical movements. This centre is probably located in the posterior portion of the second or mid-frontal convolution, or the neighbouring part of the ascending frontal.

(2) *Sensory Aphasia*.—(a) Auditory Aphasia. A lesion of the centre by which words heard are interpreted into ideas. The centre is situated in the first or superior temporo-sphenoidal convolution, and in right-handed persons is found on the left side of the brain. It corresponds to the so-called hearing or auditory centre. It probably includes the posterior third or two-thirds of the superior temporo-sphenoidal convolution. A lesion of this centre causes word-deafness and the individual so affected cannot acquire symbols by the ear.

(b) Visual Aphasia or word-blindness implies an involvement of what may be described as the higher sight centre situated in the angular gyrus which in right-handed persons is found on the left side of the brain. By this centre words or symbols seen are interpreted into ideas which can be used for purposes of speech, writing, or for being stored up in memory.

It should be remembered that there are important paths

of communication between these centres, the exact connections of which are more or less problematical, and for a full description the reader is referred to the works of Broca, Lichtheim, Wyllie, and many other writers. The position of the intellect centre is as yet unknown; very probably it includes these four centres and the neighbouring brain tissue, but whether it is unilateral or bilateral cannot at present be determined. Should a lesion of one or other of these centres occur, the corresponding area of brain on the opposite side can frequently in time be educated to take up the functions previously carried on by the damaged centre. In childhood these centres, usually on the left side of the brain, become educated largely by the eye and the ear; and in the memory centre, which probably more or less corresponds in extent to that of the intellect, the symbols indicating ideas or things are stored up.

Etiology.—Tumour, thrombosis, embolism, hæmorrhage, gumma, or abscess can involve one or more of the cortical areas, and certain of these lesions may cause damage to connecting paths. In addition it is probable that toxic agents may in some way produce temporary or permanent derangements, and it is only necessary to mention the effects of an excess of alcohol, morphia, or similar agent in upsetting the normal capabilities and co-relations of these centres with each other.

Pathological Anatomy.—In addition to the primary lesion, whatever that may be, degeneration occurs in tracts of nerve fibres severed from their trophic cells; and it should also be remembered that a comparatively small area of sclerosis may damage connecting paths between important centres without there being necessarily a lesion of the centres themselves.

The **Clinical Features** depend on the centre involved. In motor aphasia the patient is unable to speak; he may see and hear normally, and gather through the medium of both visual and auditory centres symbols and words, but he cannot give out symbols by speech. There may be the ability to utter certain words which have little bearing on what he wishes to convey, especially short words like "yes" or "no," and what might be termed explosive expressions, such as oaths, may be interjected largely as the result of reflex stimulation of centres over which the patient has practically no control. Motor aphasia is commonly associated with hemiplegia of the right side of the body, and especially where the lesion is an

extensive one in the internal capsule, or more frequently where, as the result of thrombosis or embolism, the motor areas on the left side of the brain are involved. In many cases, if the patient is shown a key and asked if it is a knife, he shakes his head to indicate that he understands but cannot pronounce the word, although sometimes if the word "key" is mentioned to him, he can automatically repeat it.

Bastian has given the name *aphemia* to cases of motor aphasia in which the lesion is in, or near, the internal capsule, because in these cases the aphasia is rarely so marked, and improvement frequently follows in a short time. This improvement is partly due to the resistance of fibres to a lesion such as hæmorrhage, but mainly to the fact that Broca's convolution is connected by an undamaged tract which crosses to the other side of the brain in the corpus callosum.

The term *paraphasia* is employed to signify the condition in which the patient uses words absolutely senseless and inappropriate.

Where the writing centre is affected, there is generally an associated paralysis of the right arm, although it should be remembered that this is not absolutely essential, because it may be communicating tracts which are involved and not the arm centre itself.

Word-blindness or visual aphasia prevents the patient from appreciating with the eye anything read, and in most cases from recognising objects or persons seen; and similarly word-deafness or auditory aphasia implies the total inability to understand words spoken. Unfortunately, as already indicated, it is extremely rare to find one of these centres alone involved, and the investigation of a case of aphasia often necessitates an elaborate and careful study of each individual centre, so as to ascertain to what extent centres themselves are affected or merely connecting paths of communication; and all sorts of peculiarities may be met with which are extremely confusing. It must also be remembered that the memory may have seriously suffered, and that amnesia or loss of memory greatly interferes with the patient's use of partially damaged centres or tracts, and adds much to the labour of the physician in elucidating the case.

Many terms have been used to indicate different lesions and their positions and groupings of symptoms due to more

complicated damage. For example, the word *apraxia* is employed to designate the inability of the patient to recognise not merely an object, but also its use and relations, but the reader is referred for a fuller account of this subject to the writings of the authorities already mentioned.

Enough has been said to show the difficulty often experienced in diagnosis, and in medico-legal cases the skill of the physician may be greatly taxed in deciding as to the amount of comprehension of which patients are capable for purposes of signing wills or other legal papers.

Prognosis.—Cases in which one centre alone is damaged may improve greatly, owing to the possibility of educating the corresponding centre on the opposite side, and it is hardly necessary to state that in syphilis the use of suitable remedies often leads to marked amelioration. On the other hand, cases of cerebral tumour and cases of extensive thrombosis are generally grave.

The **Treatment** includes what might be said in connection with the management of tumours and other causal lesions. Where one centre alone is affected, and especially in young persons, the opposite side of the brain may be capable of being trained to do the work which ought properly to be conducted by the centres on the affected side. Re-education is always slow, and even in the most favourable cases requires much patience and not a little skill.

DYSARTHRIA

By this term is implied speech defects due to lesions of the nerve cells governing the second motor neurones, their nerve fibres, and the muscles to which they go. All sorts of lesions, transient and permanent, in connection with the nose and mouth, pharynx and larynx, may interfere with the pronunciation of words, and complete inability to articulate has been termed *anarthria*. Not infrequently it is only certain letters and sounds with which the patient has much difficulty. Amongst the common types of dysarthria are slurring of syllables, stumbling over syllables, undue separation of syllables, and the reduplication of syllables, but the subject is a very wide one, and cannot be fully considered here.

3. CEREBRAL CONGESTION

(1) *Acute Cerebral Congestion*

Congestion of the vessels of the brain, and is, in certain cases, an initial stage towards actual inflammation of brain tissue; the term is often employed to describe the condition which precedes the actual rupture of the vessel in the brain in persons who have suffered from a cerebral hæmorrhage.

Etiology.—It is due to many causes, including excessive mental and physical strain, but perhaps most commonly to prolonged constipation, and it is associated with high arterial tension and arterial disease. Persons who have a high vascular tension, and who allow the bowels, kidneys, and skin to act defectively, render themselves liable to an attack of cerebral congestion. In a quite distinct class of cases excessive mental overwork may induce an attack, even in those whose vessels are perfectly healthy.

Pathological Anatomy.—The nerve cells are over-stimulated by the excessive supply of blood, and not infrequently miliary or other aneurisms are found on the arteries in persons who are predisposed.

Clinical Features.—Headache, complete unfitness for mental effort, excitement, and sleeplessness are common, and in not a few cases the phenomena suggest the probable onset of a cerebral hæmorrhage, to which, indeed, this condition is often a premonitory stage. In some cases the patient may develop a regular attack of acute mania.

Prognosis.—Recovery under proper treatment, although not infrequently repeated attacks point to the probability of cerebral hæmorrhage occurring sooner or later.

Treatment.—Absolute rest, with prompt attention to induce free action of the bowels, kidneys, and skin. Endeavour to reduce the high tension in the arteries, and curtail the dietary, especially in the direction of limiting the amount of butcher meat eaten and of alcohol consumed, and by so doing obviate the risk of future attacks in persons with arterio-sclerosis.

(2) *Passive Cerebral Congestion*

The result of any obstruction to the circulation which interferes with the return of venous blood from the brain

Headache, unfitness for mental effort, and sleeplessness are all common clinical features, and the treatment consists in endeavouring to remove the obstruction; as this is often of cardiac origin, it generally implies the administration of cardiac tonics, besides the use of purgatives and diuretics.

4. CEREBRAL ANÆMIA

Anæmia of the brain, whether from a deficient supply of blood or from the poor quality of the blood supplied.

Etiology.—It is not infrequent as the result of excessive hæmorrhage. It is due to all the causes of profound anæmia, while it may also occur in aortic incompetence and in obstruction of arteries.

Pathological Anatomy.—The nerve cells are stimulated by the want of blood, and, probably, of the oxygen it should contain; where this condition persists, the cells cease to be able to discharge their functions.

Clinical Features.—In addition to the subjective and objective symptoms and signs of general anæmia, there are the following clinical features specially referable to anæmia of the brain. These are giddiness, more or less pain in the head, sometimes tinnitus aurium, dimness of vision, dilatation of pupils, sleeplessness, and, in very marked cases, syncope. There may be definite irritation phenomena in addition to those mentioned above, as, for example, muscular twitchings, and these may be followed by depression phenomena, including paralysis, coma, and death.

The **Prognosis** depends much on the degree and continuance of the condition.

The **Treatment** must obviously be directed to the removal of the cause where this is possible, and to the supply of more and better blood to the brain.

5. CEREBRAL HÆMORRHAGE

This generally causes what is described as apoplexy, although it should be remembered that an apoplectic seizure may also be the result of cerebral thrombosis or embolism. The vessels which give way are those which are least well supported, and have to sustain the greatest blood pressure.

The antero-lateral nutrient arteries, and especially those on the left side, being in direct line with the left ventricle, are more frequently affected than are any other arteries in the brain (see Diagram 45).

Etiology.—Cerebral hæmorrhage is an affection of later life; it is more common after the age of 50, and especially in persons whose arteries are seriously degenerated. In all cases in which there is arterio-sclerosis, so often the result of alcoholism, syphilis, or overstrain, and in chronic Bright's disease, there is increased pressure in the arterial system as a whole, and the

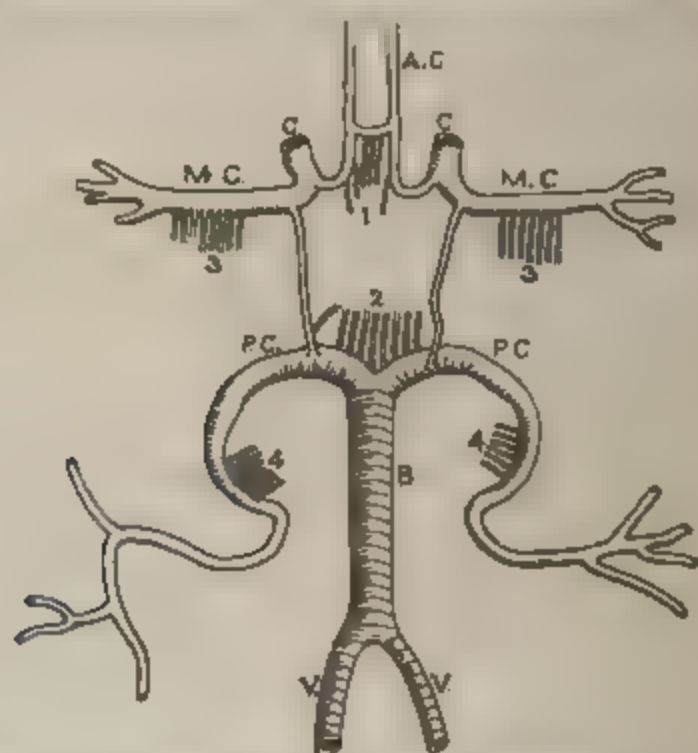


Diagram 44.—Schematic representation of the Circle of Willis, with the nutrient Arteries supplying the region of the Basal Ganglia. C. Internal carotid arteries, M.C. Middle cerebral arteries, A.C. Anterior cerebral arteries, P.C. Posterior cerebral arteries, B. Basilar artery; V. Vertebral arteries; 1, Antero-median nutrient arteries; 2, Postero-median nutrient arteries; 3, Antero-lateral nutrient arteries; 4, Postero-lateral nutrient arteries.

little nutrient arteries in the brain are apt to give way. In most cases minute miliary aneurisms form on the nutrient arteries, and it is to the rupture of one of these that cerebral hæmorrhage is generally due. Persons of what may be termed apoplectic habit, and those whose vessels have suffered in the way just described, may readily bring about the rupture of a small miliary aneurism by any unaccustomed or severe strain such as straining at stool, or the effort necessary for catching a train or train-car, or even stooping to tie a bootlace. Excessive drinking, excitement, and especially anger, may also cause cerebral hæmorrhage.

are, however, sometimes associated with specially where emboli of an infective nature into the cerebral arteries, and hæmorrhages may leucocythæmia, pernicious anæmia, purpura, and

Local Anatomy.—*The Site of the Hæmorrhage.*—*Local Hæmorrhage.*—(1) The most frequent position part of the lenticular nucleus, and the hæmorrhage

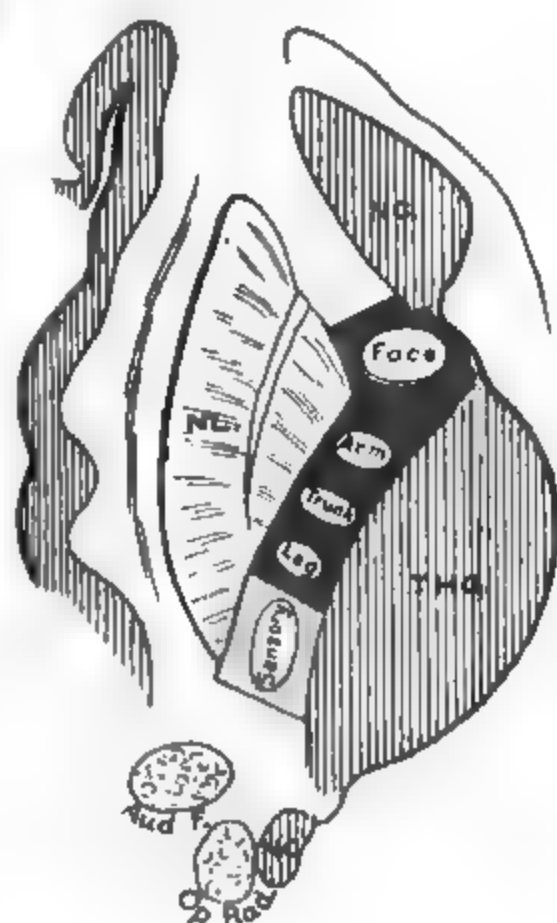


Diagram 40. —Internal Capsule, showing position of Motor (red) and Sensory (yellow) Fibres. N.C. Caudate nucleus, N.L. Lenticular nucleus; TH.O. Optic thalamus; Aud.F. Auditory fibres, Op.Rad. Optic radiation.

is due to rupture of the so-called "artery of cerebral hæmorrhage," which is one of the largest and most posterior of the lenticulo-striate arteries (see Diagram 47). The effused blood may be small in amount, and may merely press on the internal capsule, generally involving the genu or knee and the posterior half of the internal capsule, and thus causing pressure on the motor fibres,—a pressure which may be merely temporary if the blood is absorbed. In other cases the hæmorrhage is greater, the brain tissue is torn through, and the blood may burst into the lateral ventricle, or may make its way externally towards the cortex. Hæmorrhage more frequently occurs

side of the brain owing to the direct line of blood pressure between the heart and the antero-lateral nutrient arteries on that side.

(2) More rarely hæmorrhages occur from other nutrient arteries. Sometimes one of the antero-median nutrient arteries may rupture and cause hæmorrhage into the frontal lobe.

(3) Hæmorrhage may also occur into the cerebellum, and

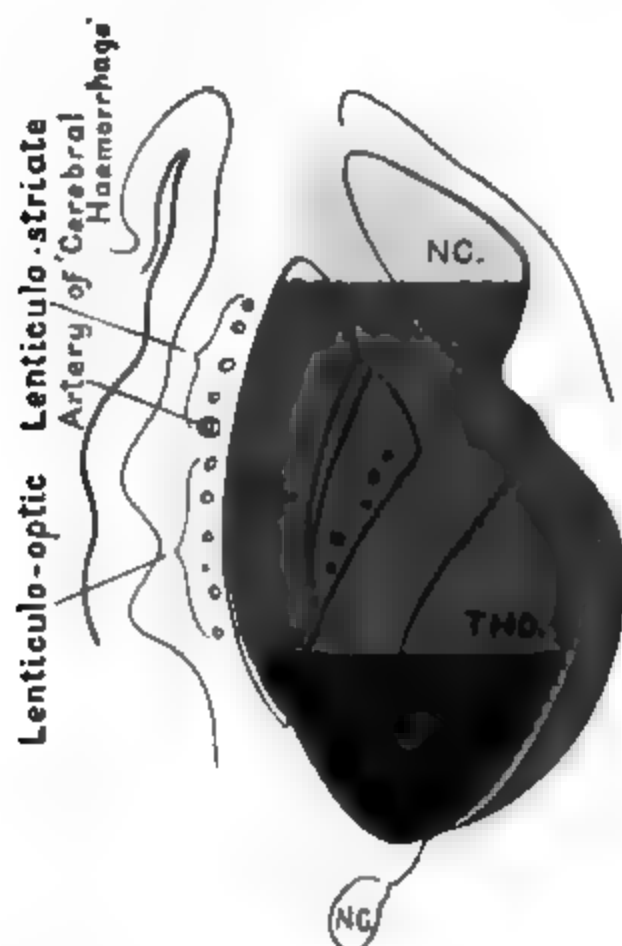


DIAGRAM 47.—Internal Capsule and Basal Ganglia, showing distribution of blood-supply by the Nutrient Arteries. Area supplied by Antero-median in yellow, Antero-lateral in red, Postero-median in green, and Postero-lateral in blue. The internal and external antero-lateral nutrient arteries are shown, and the Artery of Cerebral Hæmorrhage, which is one of the largest of the latter group.

usually proceeds from the superior cerebellar artery. It is met with in young persons generally under the age of 25, and the blood may burst into the fourth ventricle; in any case death ensues with great rapidity.

(4) Ventricular Hæmorrhage.—This is rarely of primary origin, and is usually due to hæmorrhage into the cerebral substance which has made its way into the ventricles. It may, however, occur from rupture of vessels of the choroid plexus. Ventricular hæmorrhage is said to be not uncommon in early life, and has occurred as the result of pressure on the

head during birth. The hæmorrhage may be limited to one ventricle; generally it is found in both lateral ventricles unless the channel of communication is closed by pressure, and it may fill the whole ventricular system.

(5) Small hæmorrhages scattered throughout the cerebral substance are met with in cases which may be described as toxic. They may be seen, for example, in patients who have died from hyperpyrexia of rheumatic origin.

B. *Meningeal hæmorrhage* may be secondary to intra-cerebral hæmorrhage, and the blood is very frequently subarachnoid in position, or may rupture through the pia-arachnoid and become subdural. In fracture of the skull owing to laceration of the brain, and generally as the result of contrecoup, subarachnoid hæmorrhages may occur on the opposite side of the brain from the fracture. Aneurisms of one of the larger intra-cranial arteries may rupture and cause meningeal hæmorrhage, and, lastly, meningeal hæmorrhage is not infrequent from injury sustained during labour.

The blood, once effused, tends to become absorbed, and if it is in the brain tissue, a cyst may result, surrounded by a definite wall and containing hæmatoidin pigment. This definite wall is due to secondary and localised inflammation, which is very generally a sequel to an intra-cerebral hæmorrhage. In the mildest cases a somewhat pigmented scar may alone be noted.

One inevitable result of a cerebral hæmorrhage where certain tracts of brain tissue have been damaged, is degenerative change, and therefore, where the motor part of the internal capsule is destroyed, descending degeneration occurs in the corresponding pyramidal tracts in the cord (the direct pyramidal on the same side and the crossed pyramidal tract on the opposite side from the lesion). Further, hæmorrhage in the frontal lobe may cause degeneration of fibres joining the frontal lobe with the pons, and when the cerebellum is destroyed, degeneration of the brachium conjunctivum may result. A hæmorrhage destroying the occipital lobe may produce degenerative changes in the optic radiation.

The great cause of intra-cerebral hæmorrhage is unquestionably miliary aneurisms, and it is difficult to account for their existence. They are about the size of a pinhead, and are probably associated with arterio-sclerosis; but they also depend on the fact that in brain tissue the blood-vessels are badly supported.

They are extremely common, and certainly in nearly all cases of cerebral hæmorrhage occurring after middle life these miliary aneurisms are present. The exact connection of syphilis with the presence of these aneurisms is doubtful.

The smaller multiple hæmorrhages seen in toxic conditions and also found in purpura hæmorrhagica, pernicious anæmia, and other diseases, are due to diapedesis of red blood corpuscles without actual rupture of the vessel walls.

Clinical Features.—*Premonitory Symptoms.*—Usually an apoplectic stroke is sudden, but not very infrequently the patient has suffered from what may be called *warnings*, which include a feeling of fulness in the head on stooping, ringing in the ears, or a sense of mental confusion, especially if the patient overeats or permits constipation to be present without treatment. Occasionally a localised numb feeling is felt in the limb about to be paralysed, or choreic movements may appear, called prehemiplegic chorea.

The Apoplectic Seizure.—In many cases the attack is sudden, and as a rule the patient rapidly loses consciousness, although in other cases the coma may be more gradual. There is sometimes a definite cry before the patient sinks to the ground. The face may be blue, reddish, or ashen-grey; the pupils are generally dilated, and if there is deep coma, they do not contract to any stimulus. Should the hæmorrhage irritate in some way the part of the nucleus of the third nerve containing the centre for the sphincter pupillæ muscles, then the pupils are pin-point. The respirations are typical, the breathing being stertorous, slow, and noisy, and the paralysed cheek is seen to be more puffed out than the one on the unaffected side; the pulse is found to be slow, full, and of high tension; the temperature at this time is generally normal. Although the patient is deeply unconscious, it is easy to tell which is the affected side by examining the limbs. The arm and leg on the opposite side from the lesion drop like a stone, whereas on the healthy side this absolute relaxation is not present. Often the bladder and bowels are evacuated during the attack. There are no convulsions, but on examining the eyes it is generally noted that the patient squints upwards and outwards towards the side of the hæmorrhage: in other words, he looks towards his lesion. When early rigidity or convulsions occur, the deviation may be towards the opposite or sound side.

In a hæmorrhage producing hemiplegia, we find on examining the face that it is not completely paralysed, that the muscles governing emotional movements are unaffected, but that the patient cannot quite close the eye or draw down the mouth on the affected so well as on the healthy side, and the tongue, when protruded, deviates towards the paralysed side, owing mainly to the unantagonised action of the normal genio-hyoglossus muscle. Interference with speech varies; it may be slight or very marked.

The arm is more paralysed than the leg. This is partly due to the fact that the leg muscles are supplied by fibres from both sides of the brain. The leg may even escape, and very rarely indeed is the leg more affected than the arm. In the *arm* the most affected muscles are those opposing the thumb, those rotating the arm outwards, and the openers of the hand. In the *leg*, the most affected muscles are the flexors of the leg and the dorsal flexors of the foot, and these muscles may remain paralysed long after the other leg muscles have begun to recover.

There is no muscular wasting unless from want of use, because the lesion is one of the first motor neurone, and not of the second, the trophic centres for the muscles remaining unaffected. The tendon reflexes are abolished immediately after the fit, but in a few days they become markedly exaggerated on the affected side and ankle-clonus is noted. The Babinski sign (page 604) is positive in the affected foot. The superficial reflexes on the paralysed side are diminished or unaffected.

An apoplectic shock may occur during the night, and the patient may waken to find himself paralysed on one side, or, as noted under the etiology, it may be the result of strain, such as straining at stool, or tying a bootlace, or hurrying to catch a train. In the case of a small hæmorrhage, hemiplegia may occur without any loss of consciousness, although this is certainly rare. On the other hand, where coma gradually deepens to profound unconsciousness the term *ingravescent* is applied to the condition.

Later Changes.—Generally within two days after a seizure febrile reaction occurs, and the patient, who may have wakened up out of the coma, once more becomes comatose, and may remain in this condition for a week or longer, and possibly death may occur without any improvement. The febrile reaction depends probably on inflammatory changes round the

site of the hæmorrhage, and it is during this period that what is termed *early rigidity* is apt to develop in the paralysed limbs, while trophic changes, causing bedsores, may also make their appearance. The temperature may rise to 102° or 103° F., and the patient becomes extremely restless until the more acute inflammatory manifestations have passed off.

Recovery generally begins in the leg, and the face improves before the arm, while the patient finds it possible to govern the shoulder movements before he regains control of those of the lower arm.

Late rigidity is apt to appear in muscles and limbs in which recovery has ceased; the arm may become permanently flexed at the elbow and the fingers flexed upon the hand. Flexion is the result of this late rigidity, and with the flexion there is often much pain, especially in the joints, which have become more or less rigid. The late rigidity is largely due to secondary degeneration, and associated with it there is exaggeration of the tendon reflexes. The muscles of the affected limbs which do not recover their movements tend to waste as the result of inaction, and it is remarkable how, in some hemiplegic limbs, the skin becomes glossy, the nails of the affected fingers and toes brittle, and the superficial temperature much below normal. Sometimes acute atrophy occurs, and probably this implies some involvement of the cells in the anterior horn of the cord. Amongst the more unusual results of hemiplegia are tremors and choreiform movements in the affected limbs, the latter type of movements being commonly called athetosis, but athetosis is more common in hemiplegias dating from early childhood.

Do not despair if unconsciousness should persist for several weeks, so long as it does not become deeper, because even after six weeks considerable improvement may occur in the paralysed limbs. The gait of a hemiplegic patient, if the leg does not completely recover, is very typical. As a rule the affected limb describes a semicircle, so as to prevent the toe of the boot catching on any irregularity of the ground.

Sensory phenomena are rare, and depend on the lesion involving the posterior third of the posterior limb of the internal capsule, when as a rule the hemianæsthesia is also associated with hemianopsia (from the close relationship of the fibres of the optic radiation. See Diagram 46).

One of the most distressing features associated with certain cases of hemiplegia is the mental change which may occur. A strong-minded man may become emotional like a child, and excessive irritability is frequently present in severe cases, rendering the nurse's post anything but a sinecure.

The typical description given above is that of the most common form of intra-cerebral hæmorrhage; but the phenomena resulting from a hæmorrhage depend on the site of the lesion. As already noted, if the posterior portion of the posterior half of the internal capsule is involved, hemianæsthesia results, but the hæmorrhage may be in many other positions.

If the hæmorrhage is in the crura cerebri, there may be crossed paralysis. If the artery to the nucleus of the third nerve ruptures, then the face, arm, and leg on the opposite side, and the third nerve on the same side are affected, and there is often some change also as regards sensation, while if the optic tract or the external geniculate body is involved, there may, in addition, be hemianopsia.

If the hæmorrhage is in the pons, there may be paralysis of all four limbs with pin-point pupils, hyperpyrexia, vomiting, and convulsions, as noted under cerebral localisation, or there may be crossed paralysis depending on the position of the lesion, which must in that case be unilateral (see Diagram 44). In this way the 5th, 6th, and 7th nuclei or nerves may be affected. If the hæmorrhage is in the posterior or upper part of the pons, then sugar or albumin sometimes appears in the urine.

If the hæmorrhage is primarily or secondarily intra-ventricular, and is of large amount, there is sudden and deep coma with abolition of reflexes and complete relaxation of muscles. Death is generally rapid.

If the hæmorrhage is in the cerebellum, it will probably occur in a young patient, and the clinical features will include restlessness, tetanic convulsions, and probably ataxia, and almost certainly it will terminate in rapid death.

Diagnosis.—In most cases there is little difficulty with regard to diagnosis. The evidence of arterio-sclerosis with hypertrophy of the left ventricle, and possibly a history suggesting cirrhosis of the kidneys, greatly assist in clearing up the case.

Where the onset is gradual, it is often difficult to say whether the lesion is a hæmorrhage or a *thrombosis*, but

hæmorrhage is more likely to occur when the patient's cardiovascular system corresponds to the description just given.

If the patient is comatose when seen, the lesion may be hæmorrhage, *embolism*, or *thrombosis*, and in addition there are other possibilities which must not be forgotten. These include *alcoholic stupor*, *opium poisoning*, *uræmia*, and *diabetic coma*. Embolism and hæmorrhage are generally accompanied by unconsciousness, while thrombosis, with a longer and somewhat suggestive history of premonitory phenomena, is more typically ingravescent, and not necessarily accompanied by coma. In cases of embolism we expect to find endocarditis with vegetations and the clinical phenomena associated with that disease. In thrombosis the circulation is usually enfeebled.

We should decide whether there is hemiplegia by carefully comparing the limbs and the face on the two sides. Opium poisoning produces pin-point pupils, and is simulated by a pontine hæmorrhage; but convulsions are usually associated with a hæmorrhage into the pons, and the heightened temperature is often a help. In opium poisoning remember that the breath, and very probably the contents of the stomach, may greatly assist in the diagnosis, and similarly, in eliminating alcoholic poisoning, a careful investigation should be made of any stomach contents. The urine should be tested carefully for albumin in cases in which uræmia is a possible diagnosis. It is more unlikely that diabetic coma can be mistaken, but if the urine is tested for sugar, acetone, or its derivatives, there is little chance of error.

It is important to remember that a patient may be both drunk and suffering from a cerebral hæmorrhage, and rather to err on the safe side than to risk making a definite diagnosis. In cases in which there is a subarachnoid effusion, however produced, and which has come on *suddenly*, the writer has noted retinal hæmorrhages in the eye of the affected side, and this may sometimes assist in making the diagnosis.

Prognosis.—In ordinary cases of cerebral hæmorrhage from one of the lenticulo-striate arteries much depends on the amount of blood effused, and also whether there is any recurrence of the effusion during the inflammatory or reaction stage. In favourable cases consciousness is either not lost or soon regained, and the paralysis, although possibly complete in the arm, is only partial in the leg, while within a matter of several

weeks the patient regains control over many muscles originally powerless. In these cases a slight trace of the hemiplegia usually persists, but contracture is rarely marked. On the other hand, where the patient remains comatose for a long time, or where there is almost complete paralysis of the leg as well as the arm, persisting without improvement for a considerable time, recovery can only be partial, and contracture is nearly always present and marked. Grave phenomena associated with a case of hæmorrhage are deepening coma, a considerable rise of temperature during the stage of reaction, the return of unconsciousness with this reaction stage, and the development of acute bedsores.

If the hæmorrhage is situated in the cortex, it is very often due to injury, and recovery without any contracture is probable. Infantile meningeal hæmorrhage is apt, however, to produce imbecility with spastic diplegia. Extensive hæmorrhages into the corona radiata and rupture of the blood into the ventricles nearly always prove rapidly fatal.

Treatment.—The patient should at once be placed at rest in bed, care being taken to loosen garments about the neck and chest, and if possible the head should be kept high. The next thing to be done is to ensure free evacuation of the bowels, which may readily be effected by placing on the tongue a minim of croton oil mixed with one or two drops of glycerine. Then decide whether it is advisable to perform venesection. The object of this procedure is to endeavour to lower the blood pressure, and so to arrest hæmorrhage if it be still continuing. An ice-bag may be placed over the head and hot bottles to the feet. In cases in which, from the deepening coma, the hæmorrhage is probably progressing, an attempt may be made to compress or to ligature the carotid artery, but the latter operation should only be carried out in cases in which death is imminent.

The patient will probably be confined to bed for a long time, and therefore it is wise to procure a water-bed, and to do all in one's power to obviate bedsores by carefully cleansing the skin, and endeavouring to harden any part liable to pressure. This is efficiently managed by sponging the skin with spirit after careful ablution with soap and water. Always prevent the skin from being kept moist or the bedclothes being soiled by discharges. The bladder should receive careful

attention from the first, and if necessary an aseptic catheter should be regularly passed. The affected limbs should be wrapped in wadding, and massage should not be permitted until an interval of a week or ten days has elapsed.

No excitement should be allowed, and stimulants, unless absolutely necessary, are contra-indicated. As far as possible the blood pressure should be kept low, and this can be efficiently carried out by free purgation, and sometimes by the administration of potassium iodide or tincture of aconite (m 3-5). During the stage of reaction it is specially desirable to endeavour to control the temperature, and to diminish the restlessness which may lead to further hæmorrhage, and during this stage sedatives are sometimes necessary.

After about 10 days begin with gentle massage for brief periods of time, and go on later to faradic stimulation. For late rigidity, massage and passive movements are probably as efficacious as anything else, and careful but not excessive use of galvanism. It is worth while, during the stage when the effused blood is being absorbed, to try the effect of potassium iodide in regular doses, although it is unlikely that it will cause much increase in the natural processes.

6. CEREBRAL EMBOLISM, OR EMBOLISM OF CEREBRAL ARTERIES

Etiology.—The embolus may originate from a vegetation or deposition of fibrin on either the mitral or aortic valves, or sometimes from a dilated left auricle in the appendix of which clotting has occurred. Much more rarely does the embolus proceed from clot on the wall of an artery or aneurism. The majority of cases of cerebral embolism are the result of heart disease in which vegetations occur on valves chronically affected, and in the less frequent cases of ulcerative endocarditis cerebral emboli are naturally not uncommon.

Pathological Anatomy.—Emboli more frequently enter one of the middle cerebral arteries, and generally the left. There may be a limited amount of anastomosis, provided the embolus does not obstruct an end artery, but in most cases where, for example, several branches of the middle cerebral are blocked, a large area of softening inevitably occurs. There may

be hæmorrhagic infarction owing to the part deprived of blood becoming infiltrated from surrounding vessels, or on the other hand there may be white softening, commonly called anæmic infarction. In both cases the brain substance becomes soft and diffuent. There is a considerable amount of serous infiltration into the area, and if the embolus contains supplicative organisms an abscess may develop. All shades of colour may be seen in fairly old infarctions, from brownish-red or yellow to white, depending much on the amount of, and changes in, the blood pigment present; but the typical yellow and white areas of infarction are more commonly the result of thrombosis than of embolism.

It sometimes happens that an embolus retracts, and the circulation is partly opened up into the affected territory, but on the other hand it is not uncommon to find a considerable amount of thrombosis secondary to the lodgment of a large embolism.

The **Clinical Features** depend on the vessel blocked by the clot. As a rule there is no premonitory symptom, but there should be in most cases some evidence of preceding heart disease. The onset is sudden and there may be coma, although, where a smaller vessel is blocked, consciousness may not be lost, and there is more frequently hemiplegia without coma than is the case in cerebral hæmorrhage.

If the whole of the *middle cerebral artery* is involved with most of its five branches, there is hemiplegia, and should the lesion occur on the left side of the brain, there is motor aphasia, word-deafness, and sometimes word-blindness; but the sensory forms of aphasia are more likely to occur in thrombosis than in embolism. If the *vertebral artery* is involved, it is the left branch which generally suffers, and the phenomena are suggestive of acute bulbar paralysis.

If the *basilar artery* is blocked, there is generally bilateral paralysis of the four limbs, and bulbar phenomena may also be present along with clinical features suggesting a pontine lesion.

If the *posterior cerebral artery* is blocked, there will be hemianopsia and very generally hemianæsthesia.

If the *anterior cerebral* is blocked, there may be no phenomena, or there may be hebétude with intellectual dulness.

The reader is referred to the topical diagnosis of brain lesions for a fuller account of the phenomena met with in each region. It should be stated that with the exception of the middle cerebral arteries, emboli rarely affect other cerebral vessels, and that, in them, thrombosis is by far the more common lesion.

The **Diagnosis** has to some extent been treated of under cerebral hæmorrhage, but it may be mentioned that an examination of the heart should, in most cases, demonstrate the presence of endocarditis.

The **Prognosis** is unfavourable in severe cases, and the **Treatment** must be on general lines.

7. CEREBRAL THROMBOSIS

Etiology.—It is generally the result of disease of the vessel walls, as in atheroma and endarteritis obliterans, but it may also occur in cases where the circulation becomes enfeebled, as in old age or in typhoid fever, and in many cases there is a combination of marked atheroma together with debility.

Pathological Anatomy.—A description of infarction has already been given under the head of embolism. It is probable that in thrombosis the anastomotic supply of blood to the affected area will not be so free as in embolism, and the patch of softened brain is usually yellow or greyish-white in colour and milky in consistence. Hæmorrhage into a patch is not so common as in embolism.

All arteries of the brain may be involved by thrombosis, and in many cases numerous small patches of softening occur which may be in silent areas, before a more important part of the brain is affected, giving rise to definite paralytic phenomena. In the brains of old persons, yellowish patches of softening, commonly called "*plaques jaunes*," may be seen in the grey matter, and as many as 20 or 30 may be counted in a case in which no very serious clinical phenomena have resulted from their development.

Clinical Features.—There are often premonitory symptoms, such as vertigo or giddiness, and headache, and very frequently before an important area of brain is affected, the patient may have experienced numerous attacks of giddiness without losing

consciousness or experiencing any other trouble. Sometimes the patient, if walking on the street, may have to hold on to the railings, or even sit down for a few moments before he feels better.

Where a serious thrombosis occurs, there are generally repeated attacks which lead up to the more extensive involvement of vessels. There may be gradually developing hemiplegia, one limb being affected after another, sometimes with a transient improvement in the affected limbs, in other cases without any marked recovery of movement. Where a large vessel is blocked, such as the whole of one of the middle cerebral arteries, coma supervenes. The attack may occur during the night, and the patient awakens to find that he is hemiplegic, and if the lesion be on the left side of the brain, he may have motor aphasia, or some form of sensory aphasia.

Where the lesion is acute, by which is meant that a large vessel is suddenly and completely blocked, the condition closely resembles cerebral hæmorrhage, and in all cases in which there are repeated attacks, or a large vessel becomes thrombosed, the patient's memory and mental powers suffer seriously.

Diagnosis.—A careful examination should be made of the condition of the peripheral vessels so as to ascertain the amount of atheroma present, and a history of syphilis is suggestive of endarteritis obliterans. There are cases in which it is extremely difficult to differentiate between thrombosis, *hæmorrhage*, and *embolism*, but in most cases of thrombosis in older patients there have been numerous previous attacks of milder nature, while in hæmorrhage and embolism the attack is more sudden and unexpected. In thrombosis, the patient rarely loses consciousness unless a large vessel is thrombosed. In hæmorrhage, consciousness is generally lost, while in embolism, coma more frequently supervenes than in thrombosis.

An attempt should be made in every case to decide with regard to the diagnosis, because the treatment for hæmorrhage is entirely different from that for thrombosis.

The **Prognosis** is bad in serious cases, because repeated attacks are apt to occur, and it is only in mild cases, or in the cases which occur as the result of the debility associated with typhoid or other fever, that a fairly favourable prognosis is warranted.

The reader is referred to the topical lesions for a fuller account of the phenomena in each region. It should be stated that with the middle cerebral arteries, emboli rarely occlude the vessels, and that, in them, thrombosis is a common lesion.

The **Diagnosis** has to some extent to be based on cerebral hæmorrhage, but it may be confirmed by examination of the heart should it be found in the presence of endocarditis.

The **Prognosis** is unfavourable.

Treatment must be on general lines.

7. CEREBRAL THROMBOSIS

Etiology.—It is generally due to changes in the vessel walls, as in atheroma and arteriosclerosis, but it may also occur in cases with no such changes, as in enfeebled, as in old age or in cases where there is a combination of atheroma with debility.

Pathological Anatomy.—The changes in the vessel walls have already been given under the heading of atheroma. It is probable that in thrombosis the blood supply to the affected area will not be maintained, and the patch of softened brain is pale in colour and milky in consistency. Thrombosis is not so common as in embolism.

All arteries of the brain are affected, and in many cases numerous small arteries are involved, which may be in silent areas. The area of the brain is affected, and the phenomena are similar to those of softening. In the brains of cases of softening, commonly called "arteriosclerotic," the changes in the grey matter, and as seen in a case in which no very marked changes resulted from their development.

Clinical Features.—There are no special features such as vertigo or giddiness, or any other, before an important area of the brain has been affected. They have experienced numerous at-

The internal jugular vein may be empty of blood. In septic cases, the whole side of the neck is swollen and painful, while there is delirium and coma, muscular twitchings, and sometimes optic neuritis may be present.

Thrombosis in any sinus is of pyæmic nature, and abscesses will be met with in the lungs and other organs. The clinical features are pyæmic in type.

In simple primary cases, rest in bed with elevation of the head is indicated, and probably an attempt should be made to prevent obstruction to the flow of blood in the sinuses by keeping the head almost flat in bed, in order thereby the occlusion of vessels due to the pressure of the head may be prevented. Stimulants such as brandy and alcohol may be given, and in certain cases iodide and mercury have been recommended.

9. ANEURISM OF THE CEREBRAL ARTERIES

This does not include miliary aneurisms, but refers only to aneurisms generally found on one of the branches of the circle of Willis.

Ætiology.—It is not common. It is more frequent in men than in women, and generally at an age when aneurism tends to develop. It is due to weakening of the arterial wall, and may be associated with syphilitic disease or with endarteritis, atheroma, or embolism. Osler's list of 22 cases includes 1 on the internal carotid, 5 on the middle cerebral, 3 on the basilar, and 3 on the anterior communicating. In other statistics nearly one-third of the cases have been found on the middle cerebral. In cases seen post mortem by the writer, the anterior cerebral was involved by aneurisms about the size of a large pea.

Clinical Features.—The aneurism may rupture and so cause death, or it may act as a tumour and press upon various important structures. It may give rise to optic neuritis, or to paralysis of some of the cranial nerves, and as a rule its termination, where death is due to the aneurism, is by rupture. Occasionally audible murmurs may be heard on ausculting the skull, but in most cases the clinical evidence of the

existence of an aneurism depends on interference with cranial nerves.

10. INTRA-CRANIAL TUMOURS

Tumours may develop from the brain, from the membranes, or from the bones of the skull, while aneurisms and cysts may produce the same symptoms as ordinary intra-cranial tumours.

(1) *Gliomata* are probably the commonest tumours originating in the brain substance itself. They develop from the neuroglia, and usually infiltrate the cerebral tissues having, therefore, no definite wall, and in process of their growth a long time may elapse before the nerve fibres passing through the tumour area lose their function.

(2) *Sarcomata* more commonly develop from the membranes, and sometimes from the bone or periosteum of the skull. This is one of the largest and most rapidly spreading intra-cranial tumours, and numerous nodules may be found throughout the brain substance.

(3) *Tubercular tumours* vary greatly in size, and are most commonly found about the base of the brain or in the cerebellum. They may be single, but more generally are multiple, and in size vary from a pea to a walnut. They are frequently met with in young patients, are apt to undergo degenerative changes, and have almost always a sharply defined outline.

(4) *Chemata* are generally superficial, and are usually attached to the membranes: they are often single, but may be multiple, and as they sometimes involve the region of the pons, both 3rd nerves are apt to be affected.

(5) *Carcinoma* is very rarely of primary origin in the brain, but secondary tumours may be met with where the primary lesion is in the breast or some other part of the body.

There are many other tumours, amongst the most interesting being *psammomata*, which are probably of sarcomatous nature. *Endotheliomata*, *epitheliomata*, and other rare tumours, simple and malignant, are occasionally met with. *Histiocytomas* and the *gliosarcomata* likewise are sometimes found in the brain, but more frequently in foreign countries.

Cysts, the result of hæmorrhage and *porencephaly*, which is sometimes of developmental origin, may cause certain of the phenomena suggestive of tumour.

Clinical Features.—The following typical phenomena are met with in cerebral tumour:—

General Symptoms.—(1) Headache, dull and aching, or sharp and lancinating, and sometimes indicating the site of the tumour. Not infrequently, when the tumour presses on some sensory nerve, there are neuralgic pains shooting along the line of the branches affected.

(2) Cerebral vomiting is a typical clinical feature. By the term cerebral is meant vomiting not directly connected with the ingestion of food, but often associated with a special paroxysm of headache.

(3) Vertigo or giddiness, varying considerably in degree, is sometimes marked when the patient rises from the prone position or when he turns suddenly when standing up.

(4) Optic neuritis occurs in four-fifths of all cases of cerebral tumour. It is almost constantly present in cases in which the tumour is situated in the cerebellum, or when it involves the corpora quadrigemina, while it is less common in cases in which the tumour is limited to the pons or medulla. It is more frequent where there is a glioma, sarcoma, gumma, or cerebral cyst, and it is less common in tubercular tumours.

Other less frequent phenomena are slowness of pulse and sometimes respirations, convulsive seizures, fainting attacks, wasting and bodily weakness, and occasionally mental manifestations, which are often very typical. As the tumour enlarges, the patient tends to become dull, sleepy, and slow of thought and speech, and the memory may fail. It is much more uncommon to find mental excitement even of a transient nature.

Focal Symptoms.—The reader is referred to the description already given of cerebral localisation, p. 640. In every case of supposed cerebral tumour the calvarium should be palpated and percussed with the finger, because, where the tumour is superficial, a considerable amount of pain is elicited on pressure over it.

It not infrequently happens that the tumour presses on the veins of Galen, causing distension of the ventricles of the brain. This is more likely to occur where the tumour is

situated near the basal ganglia. As a result the evidences of pressure on the cerebral cortex and the important centres in the brain become more marked, and eventually the irritation phenomena, which include vomiting, giddiness, and headache, are replaced by coma and slowing of the pulse, suggestive of the paralytic stage being reached. There is no doubt that choked disc is more common where there is great intra-cranial pressure, but it is impossible in every case to deduce from the examination of the eyes satisfactory evidence of pressure on the veins of Galen. In children there is sometimes definite separation of the bones of the skull, but in the adult this is almost impossible. The tumour, if near the base of the brain, frequently presses on one or more of the cranial nerves, and this may aid in its localisation.

Towards the termination of a case of cerebral tumour there is usually greatly increased intra-cranial pressure resulting in coma, which may, however, be relieved by operation undertaken for the purpose of reducing the tension within the skull.

Diagnosis. — We have to decide whether there is a tumour, and if so, what is the nature of the tumour and where it is situated. The presence of the general phenomena suggesting cerebral tumour, together with any evidence indicative of its site, helps greatly in the diagnosis. The *albuminuric retinitis* met with in Bright's disease differs considerably from the papillitis usually seen in cerebral tumour, and there is no particular reason why, in cerebral tumour, albumin should be present in the urine unless the tumour involves the region of the 4th ventricle. Sometimes cases of *general paresis of the insane* closely resemble cerebral tumour, but the history of the case and the absence of optic neuritis should, as a rule, prevent mistake, and in *hysteria* it is rare to find the group of cardinal clinical features so commonly met with in intra-cranial tumour.

We must depend on the knowledge we possess of the functions of different parts of the brain to aid in the diagnosis of the position, but with regard to the nature of the tumour we are often greatly at a loss. In younger patients tubercle, but also glioma and sarcoma are common, and the diagnosis favours glioma or sarcoma if optic neuritis is present. In older patients, and those with a syphilitic history, gumma is by no means improbable.

The **Prognosis** is extremely unfavourable, with the exception of syphilitic cases, and those in which the focal phenomena are very distinct and suggest cases which are operable. Many patients survive for years if the tumour is chronic, but the more probable duration is a matter of months.

Treatment.—We should invariably commence with potassium iodide and mercury, and the former, at all events, should be pushed, if the smallest benefit seems to result. Sedatives are often of value; bromides, chloral, and opium may all be given, and ice frequently helps to ward off the sickness. Rest in bed adds much to the comfort of the patient, and in certain cases where vomiting is very severe, feeding by the bowel may have to be resorted to. An ice-bag applied to the head relieves the headache.

The question of operation should invariably be thought of. It is desirable, even in cases in which there are no definite localising phenomena, but in which optic neuritis is threatening to render the patient blind, to trephine the skull although it be merely to relieve pressure. There is certainly the risk that, after trephining, a *hernia cerebri* may result and give rise to trouble, but where pressure is rapidly increasing and the headache becomes unbearable, trephining should not be delayed. In a number of cases where the tumour has been localised, the result of operation has been satisfactory, and the improvement with regard to sight has often been remarkable and encouraging.

11. ENCEPHALITIS

The term implies inflammation of the brain, which may be acute or chronic, a somewhat trivial hyperæmia, or a serious and fatal infective suppuration. Many forms of encephalitis are certainly of toxic origin, and, as stated in the descriptions of acute bulbar paralysis and infantile paralysis, the lesions in these diseases are exactly similar to toxic encephalitis. There are also many forms of encephalitis due to direct infection of brain tissue as the result of fracture of the skull, infection from a cerebral abscess, caries of bone and infective thrombosis, while in ulcerative endocarditis and pyæmia there may be numerous small capillary emboli containing organisms and capable of setting up a corresponding number of local areas of cerebritis.

The affected brain tissue varies considerably in appearance, depending much on the amount of hæmorrhage associated with the inflammation and the presence or absence of pus. In many forms of encephalitis the pathological changes are limited to hyperæmia with a certain amount of inflammatory œdema, while in other cases actual softening and even suppuration may result.

The **Clinical Features** depend on the type of inflammation present, but headache, delirium, convulsions, and eventually coma are extremely common, while all the symptoms included under the term *malaise* are generally present. The earlier phenomena due to the cerebral condition are *irritative*, and those which develop later are *paralytic*. Probably many cases of concussion of the brain are in reality encephalitis due to traumatic injury, and not infrequently in a severe case where recovery occurs, the mental faculties may be seriously interfered with, either temporarily or permanently.

Various names have been given to encephalitis in different parts of the brain: for example, superior polio-encephalitis, described by Wernicke, involves specially the nuclei of the ocular nerves, whereas inferior polio-encephalitis is the term given to cases in which the other cranial nerve nuclei in the pons and medulla are affected. The clinical features present will depend on the nuclei involved, and the chief phenomena are referred to under bulbar paralysis.

The **Diagnosis** is often difficult, and the disease resembles, as regards clinical history, a case of infantile paralysis beginning with a definite febrile attack.

The **Prognosis** depends much on the site and severity of the lesion, and the **Treatment** should be on general lines, special attention being paid to rest in bed, to purgation, to the administration of sedatives such as the bromides, and in certain cases to the use of iodide of potash and mercury.

CHRONIC ENCEPHALITIS

It is possible that any lesion giving rise to encephalitis may induce a chronic rather than an acute condition. The clinical features are similar, but less severe.

It has been suggested that chronic encephalitis may occur as the result of alcohol or gonorrhœa.

12. CEREBRAL ABSCESS

An encephalitis of suppurative nature, and which may be found in the brain tissue itself or between the membranes and the brain.

Etiology.—Cerebral abscess may be the result of injury, and especially fracture of the skull, the abscess being found in close relationship to the site of the lesion; foreign bodies, such as bullets, sometimes carry into the brain septic organisms which may set up suppuration.

Disease of the middle ear is probably the commonest cause of abscess in the brain, and the abscess may be secondary to a septic thrombosis of the lateral sinus infected from the ear. It generally results from caries of bone, by which in time the meninges become involved. The most common site of perforation is the roof of the tympanum, but the pus may find its way along any of the lymphatic or other channels of communication between the ear and the inside of the skull. Very rarely does suppuration of the outer ear extend by the lymphatics to the brain without the middle ear being involved. In cases of abscess secondary to ear disease, the abscess is often found in the temporo-sphenoidal lobe, and rather less frequently in the cerebellum.

Sometimes in diseases of the nose, caries of the ethmoid bone occurs, and in this way the brain may become involved. The abscess is most frequently in one of the frontal lobes and in close proximity to the affected bone.

Very rarely does suppurative disease of the orbit extend to the brain, and it is only occasionally in cerebral tumours that pus formation occurs, perhaps most frequently in tubercular cases.

Cerebral abscesses may be the result of pyæmia, and they are very common in cases of ulcerative endocarditis. In bronchiectasis, and more rarely in other conditions, metastatic abscesses have been found in the brain.

Abscess of the brain is sometimes a sequel to a continued fever, and there are cases in which a cerebral abscess has occurred without any recognised primary cause.

Pathological Anatomy.—A cerebral abscess is generally single, and varies considerably in size. The pus is often stinking in odour and greenish in colour, while there is usually

in the less acute cases a definite capsule. Depending upon the size of the abscess, there is more or less flattening of the cerebral convolution, and in some cases suppurative meningitis is present.

The organisms most frequently found in cerebral abscess are Staphylococci and Streptococci, although many others may also occur.

The **Clinical Features** vary greatly in different cases; sometimes there is undoubted evidence, owing to the irritation phenomena present, that a cerebral abscess exists. In other cases there may be no symptoms at all, or, at all events, the symptoms present may not suggest the existence of an abscess. There may be headache, pain sometimes increased on tapping over the site of the abscess, cerebral vomiting, marked vertigo, and occasionally optic neuritis, and these may all be described as *irritation* phenomena. The pulse is usually slow, it may be only 40 to 50, and the temperature is often subnormal; the respirations are slow, although sometimes they assume the Cheyne-Stokes rhythm. Focal symptoms of cerebral abscess are of the utmost importance, and depend on the site of the pus. The most careful examination should be made of the ears, of the skull, and of the nose, so as to endeavour to find out a possible cause for a supposed abscess of the brain. The reader should refer to the description of the phenomena which characterise a cerebellar lesion (p. 647), although it must be remembered that a cerebellar abscess may occur without any typical phenomena whatsoever. MacEwen considers that the percussion note obtained over the skull becomes more resonant when the lateral ventricles are distended with fluid, and this may be of assistance in a case of cerebral abscess.

The **Diagnosis** is often of very great difficulty, especially where there are no focal symptoms and no definite evidence of the presence of an abscess at all. It is wise to investigate the blood so to determine the presence or absence of a leucocytosis, and the optic discs should be examined with the greatest care. In the writer's opinion too much stress is laid on the supposed frequency of optic neuritis in cerebral abscess.

In *thrombosis of the lateral or other sinuses* of septic nature, a careful examination should be made of the neck, and also of any swelling over the mastoid region, the position of the

mastoid vein which enters the lateral sinus being kept in mind.

The **Prognosis** depends on the possibility of successful operation, and on the probable absence of meningitis or of an extensive area of encephalitis.

The **Treatment** is purely surgical, and is therefore omitted. It is important to remember that early operation is desirable, and that opium or morphia should not be given where important symptoms might be masked by its use.

13. CHRONIC HYDROCEPHALUS

The term generally implies distension of the ventricles of the brain with cerebro-spinal fluid, although cases in which there is an excess of fluid between the dura mater and the brain have also been included, and the terms internal and external chronic hydrocephalus are accordingly used to distinguish the two varieties.

(1) Internal Chronic Hydrocephalus

Etiology.—It may occur in the fetus and interfere with normal labour, or it may develop after birth; in both cases it is probably due to inflammation of the ependyma ventriculi. In certain instances this inflammation may be of tubercular origin, but in others there is no satisfactory explanation obtainable.

In adults this form of chronic hydrocephalus is usually due to pressure on the veins of Galen by a tumour, or to occlusion of the foramen of Magendie; but sometimes inflammation of the meninges or of the ependyma accounts for the condition. It may be associated with spina bifida or syringomyelia.

Pathological Anatomy.—Where there is great distension of the ventricles, the brain is greatly thinned, the convolutions are flattened, and the sulci are almost obliterated. There may be from 5 to 20 pints of fluid in the lateral ventricles, and the bones of the skull are widely separated, while the appearance of the child's head described below is extremely typical. The fluid is generally clear like water, containing a trace of albumin, and the ependyma lining the ventricles is much

thickened, opaque, and covered with greyish granulations. The presence of a tumour may be noted where it accounts for the condition. Sometimes the pressure is so great that the optic commissure is flattened by the distended third ventricle.

Clinical Features.—The head is greatly enlarged, and in the child may measure 30 to 50 inches round the forehead. The frontal and temporal regions appear to overhang the small, pinched-looking face. The bones of the skull are widely separated, and *ossa triquetra* or Wormian bones may form in the gap between the bones or over the fontanelles. The head may be so heavy that the patient is unable to raise it from the pillow, or he may have to support it between the hands. There may be few compression phenomena, or there may be blindness due to pressure on the optic commissure. The intelligence varies greatly: it is sometimes deficient, or it may be normal; but it is interesting to note that in the child, if ossification of the bones is nearly completed, the pressure phenomena are more marked, and therefore severe headache and vomiting are extremely common. Nystagmus is frequently present in severe cases in children.

In adults the head cannot so easily enlarge, and in consequence the evidences of pressure are much more serious. Headache, vomiting, convulsions, and paralysis due to pressure on cranial nerves, are common, and certainly in cases of tumour optic neuritis and atrophy frequently develop.

Diagnosis.—In the young child a glance at the head is sufficient to render the diagnosis clear, while the patent fontanelles found in older hydrocephalic children will help in any doubtful case.

In adults it is usually easy to recognise increase of intracranial pressure, although the cause of that increase may be obscure.

Sometimes cases occur in which there is enlargement of the head commencing after adult life, and probably similar in nature to acromegaly, but this is extremely rare.

The **Prognosis** is unfavourable, although in not a few instances benefit is obtained by careful treatment. In some cases, after tapping, the fluid does not reaccumulate rapidly, while in other cases the ventricles fill up in a very short time. In tumour cases the possibility of removal of the neoplasm pressing on the veins of Galen is extremely problematical.

Where there is chronic meningitis present, the prognosis in adults is more favourable, but even in these the percentage of recovery is extremely small.

Treatment.—The historic case in which a child with hydrocephalus fell down, struck its head upon a nail, which perforated the skull and opened into one of the ventricles, is certainly encouraging, because in that case recovery occurred, and fortunately without sepsis developing.

In young children it is wise to endeavour to tap the ventricular effusion. This may be done in different ways, either—(1) by lumbar puncture, when, provided there is free communication, the excess of fluid can be greatly diminished; or (2) a child whose anterior fontanelle is still patent may have the operation performed by inserting the trochar and canula at the outer angle of the fontanelle, care being taken to remove only a few ounces at a time; or (3) the skull may be trephined and the fluid removed either from time to time or by continuous drainage. This operation is obviously more suitable for adults. Watson Cheyne recommends an attempt at making permanent drainage into the subdural space.

In children, after tapping by one or other of these methods, the head should be compressed either by an elastic bandage or strapping, but care should be taken to prevent the appearance of either cerebral irritation or paralysis due to too great pressure. A course of diuretics and purgatives has sometimes been found useful, and the patient's health may be benefited by the administration of nourishing food and tonics.

(2) **External Chronic Hydrocephalus**

In this condition the fluid accumulates outside the cerebral convolutions, and is generally due to atrophy of the convolutions, although it may also depend upon meningitis. The cerebro-spinal fluid is found in excess in cases of general paralysis of the insane, and in all cases in which the brain either does not develop properly or where it atrophies.

The **Clinical Features** are those suggestive of atrophy of the brain, the intellectual functions usually failing, while in not a few cases the typical manifestations of general paralysis appear. In idiots in whom the brain does not develop at the same rate as the skull, there are often no particular clinical features due to the presence of the fluid. Little can be done

by way of treatment in these cases. In general paralysis it may be desirable to trephine the skull so as to relieve pressure.

14. MULTIPLE CEREBRO-SPINAL SCLEROSIS

(DISSEMINATED SCLEROSIS—INSULAR SCLEROSIS)

A chronic disease affecting the brain and cord, in which patches of sclerosis replace the nerve elements; they may be situated in practically any position.

Etiology.—It is a very difficult matter to assign the proper causes to this disease. In some cases there seems little doubt that cold and exposure, injury, excitement, and sometimes an acute infective fever, are responsible for the condition. It is common in both sexes, it occurs soon after puberty, and also in middle life, and it seems not improbable that it may really be of infective origin. It should be stated that many authorities attribute to syphilis an important causal relationship.

Pathological Anatomy.—The patches of sclerosis vary greatly in size, they are not limited to any particular tract, and the same patch may involve white and grey matter; they are pinkish in appearance, but when older they become greyish-white, and they are often hard and cartilaginous to the touch. The patches are specially common in the region of the basal ganglia, in the pons, medulla, and cerebellum, while in other cases the cord, and particularly the cervical region, may contain several of these areas. The microscopic structure of the patches is simply a great proliferation of neuroglia forming a dense, firm nodule, which eventually destroys the nerve fibres or cells in the affected region, although it is a remarkable fact that for a long time the axis-cylinder processes persist, and may, in an unsatisfactory way, discharge their functions. In consequence of this, less secondary degeneration results than might be expected. It seems probable that the sclerosis is toxic; whether due to an organism or not we cannot definitely say.

Clinical Features.—The disease is chronic, insidious, and gradual in its development, and the phenomena may be divided into three stages, of which the first is the earliest, and the last terminates with the death of the patient. These three stages have been described by Charcot, who considers that the *first* stage terminates with the disablement of the patient, due to spastic rigidity of muscles; that the *second*

beginning with the rigidity, terminates with the loss of the organic reflexes; and that the *third* stage, beginning with the loss of these reflexes, terminates with bedsores and death. The stages may be long protracted, and there is no given time when the patient should pass from one stage to the next. Usually the disease begins with loss of power in the legs, and the patient complains of feebleness in movement, with gradually increasing stiffness and rigidity amounting to spastic paralysis, and associated with this there is a marked increase in the tendon reflexes.

There are three important clinical features which are typical of the disease.¹

1. *Intention Tremors*.—These are peculiar involuntary quick jerks observable on voluntary movements. They generally appear first in the arms or in one arm, are markedly increased by nervousness, and are not under the control of the patient. The more the patient endeavours to arrest the tremors the worse they become, but they cease entirely during sleep. If the patient is given a glass of water to drink, he tries to raise it to his lips, but the jerky, uncontrolled oscillations, which increase as he continues the movement, make him spill the bulk of the contents of the tumbler, and as his head participates to some extent in the same kind of movement, the tumbler may be brought with a sudden dash against the patient's teeth; once he begins to drink, his arm becomes much steadier.

Some loss of power accompanies this tremor, but the legs generally show more weakness with spastic paralysis than loss of co-ordination. The marked development of this spastic paralysis with increased knee-jerks and ankle-clonus constitutes the rigidity described by Charcot as terminating the first stage of the disease.

2. The *speech* becomes *scanning*, *staccato*, and *syllabic*, with slow, monotonous, stammering articulation, the ends of the words are cut short, and along with this peculiarity, serious lapses of memory and very defective intelligence are associated.

3. *Nystagmus*, generally lateral and rarely vertical, is almost always present on moving the eyeballs to one or other side.

Headache and giddiness are extremely frequent, and are

¹ It is remarkable how many atypical cases present themselves, and how often the distinctive intention tremors are absent. This depends on the site of the patches.

often bitterly complained of by the patient. The mental conditions have just been mentioned, defective intelligence and the almost complete failure of memory being common, and sometimes insanity develops in the course of the disease.

The other clinical features vary, depending on the sites of the patches of sclerosis. The optic nerves may be affected by a patch involving the chiasma and causing secondary optic atrophy, and paralysis of some cranial nerve may result from a patch involving its nucleus; in this way the eye muscles may not infrequently be paralysed.

Sometimes patches of anaesthesia occur, although ordinary sensation is not, as a rule, much affected.

In the last stages of the disease the loss of control of the organic reflexes and the bedsores rapidly lead to a fatal result.

Diagnosis.—The three great clinical features are very typical; it must, however, be remembered that we may find disseminated sclerosis associated with other diseases, such as locomotor ataxia and general paralysis of the insane.

The **Prognosis** is bad: the patient will probably live five or six years; in some cases the first stage may be even more protracted, and the question is rather the rate of progression downwards than anything else. In favourable cases the disease may be arrested for a considerable period of time.

Treatment.—Antisymphilitic remedies should certainly be tried; arsenic and silver nitrate have been commended, but no drug seems to be of much avail. Recently hydropathic and electrical treatment, and rhythmical exercises on the Swedish system, have received considerable attention, but the results are very disappointing.

15. INFANTILE HEMIPLEGIA

A small percentage of all cases of hemiplegia are found to be of congenital origin. Both sexes suffer equally, and inherent delicacy does not appear to be so important a factor as one might imagine. Opinion has varied greatly as to whether alcohol and syphilis in the parents have much to do with this form of hemiplegia in the child. Of the causal factors with which we can speak with certainty, injury at birth is unquestionably the most frequent, the head being compressed by the forceps in cases of instrumental delivery. In other cases, the hemiplegia

develops during the early years of life, and probably infectious diseases are responsible for a large number of these cases. A convulsive seizure precedes the paralysis, and on recovery the hemiplegia is noted.

Pathological Anatomy.—In cases occurring at child-birth hæmorrhage or injury to the cortex of the brain produces the condition. Atrophy of the brain cortex, whether the result of meningeal hæmorrhage or not, is associated with a large proportion of all cases of congenital hemiplegia. Sometimes there is more or less distension of the ventricles. A cystic formation, commonly called porencephaly, has been found present in a number of cases. These cavities are most probably of developmental origin; some of them are connected with one of the lateral ventricles.

In a small proportion of cases hæmorrhage, thrombosis, and embolism may be present, similar in all respects to these conditions in the adult.

Clinical Features.—There are generally one or more convulsions, followed by the development of the hemiplegia, which is noted when the child recovers consciousness; but, as the pathological anatomy suggests, there are also cases in which the paralysis develops more slowly—cases in which atrophy or arrested development of one-half of the brain is present.

In some instances the hemiplegia gradually disappears; in others it persists more obviously in the arm than in the leg, and contracture is by no means infrequent, while athetosis is peculiarly common in these cases. The athetoid movements are slow, rhythmical, and involuntary flexion and extension movements of the fingers and thumb, while hyper-extension occurs, and the fingers are widely separated; the movements are increased by emotion, and may persist more or less during sleep. Probably the feature which is most striking in addition to the athetosis consists in the large number of cases in which mental weakness is present, and all stages and types of imbecility may be found in these patients. Epilepsy is also a fairly frequent sequel.

The **Diagnosis** is not difficult. The lesion is one of the first motor neurone, and therefore the muscles do not waste unless from disuse; it is easy to distinguish between a lesion of the cells in the anterior horn of the cord in *infantile paralysis*, because in the latter case wasting is extreme, and

there is abolition of reflexes, and also loss of electrical reactions ; whereas in infantile hemiplegia not merely is there less wasting, but the reflexes tend to be exaggerated, and the electrical reactions are fairly normal.

The **Prognosis** is unfavourable in most cases.

The **Treatment** is most unsatisfactory. Where a hæmorrhage has just occurred under the membranes, it may be possible by surgical interference to do something for the patient, and sometimes good feeding and care, with massage to the affected limbs, may result in great improvement in a case which, at first sight, appears hopeless. Imbecility and epilepsy are very unfavourable clinical features, and little can be done surgically in such cases, while the later treatment consists in the careful training and education of the child.

16. INFANTILE DIPLEGIA

(LITTLE'S DISEASE—SPASTIC DIPLEGIA)

These cases almost entirely originate from injuries resulting at birth, only both hemispheres are involved in place of one, and generally hæmorrhage under the membranes is the chief lesion, the brain being compressed by a meningeal blood-clot.

The **Clinical Features** include convulsions, weakness of the arms and legs, with typical rigidity and spasticity. Very frequently there are constant choreiform movements of the affected limbs, and the child is deficient mentally. The face muscles show similar irregular movements. In a small proportion of cases the child, although bodily affected in this way, is mentally intelligent.

The **Prognosis** is unfavourable owing to the inability to do anything to relieve the pressure on the brain at the time of the injury, although recently surgeons have successfully operated in a number of cases, and such treatment is the only thorough and satisfactory procedure.

Should the patient be seen after years have elapsed, little can be done to limit the movements, or to aid him in using the limbs.

17. GENERAL PARALYSIS—DEMENTIA PARALYTICA

A primary degeneration of the brain, with **progressive** motor paresis and dementia.

Etiology.—The supposed causal factors include:—syphilis acquired or inherited, or some unknown toxin; worry, mental overstrain, or continued excitement, and possibly physical overwork; alcoholism; sexual excess; blows on the head and sunstroke. Probably a combination of some of these causes exists in most cases. Males of adult age suffer chiefly, and the disease is rare after 50.

Pathological Anatomy.—The brain is markedly atrophied, especially anteriorly, and there is a corresponding excess of cerebro-spinal fluid; the lateral ventricles are enlarged, and the ependyma is thickened. The cerebral membranes are also thickened, and pachymeningitis interna hæmorrhagica may be present. The neuroglial cells of the cerebral convolutions are greatly hypertrophied, and have undergone proliferation, and the cortical nerve cells show marked degenerative changes with chromatolysis. The cord is similarly affected, but to a less degree. The perivascular lymphatics are much dilated, and there is an increase of mononuclear cells.

There is some difference of opinion as regards the disease, whether it is a meningo-encephalitis or a primary degeneration of the cerebral cortex, the neuroglial proliferation being secondary. It is very probably of toxic origin, and according to Ford Robertson is dependent on the presence of diphtheroid bacilli.

Clinical Features.—No disease possesses so many different symptoms or types. The leading features common to all cases are:—(1) Progressive mental changes, and especially dementia with excitement or depression. (2) Progressive motor weakness with tremor, chiefly of the tongue and face. (3) Hesitating, stammering, jerky speech, often with dropping of syllables, while labials, linguals, and dentals become difficult, such words as “British constitution” and “artillery” being specially troublesome to the patient. (4) Writing is affected soon after speech, and words or syllables are omitted, while much confusion of ideas exists. (5) The pupils are unequal, and usually show loss of the light-reflex (the Argyll Robertson phenomenon) while reacting to accommodation. (6) The knee-jerks are generally affected, in certain cases exaggerated, in others absent, while sometimes they differ on the two sides.

The tremor of face muscles is well seen on asking the patient to show his teeth, and the fingers are often tremulous. Optic atrophy may be present in a small number of cases,

and sometimes paralysis of oculo-motor muscles, temporary or permanent.

Apoplectiform or epileptiform attacks are not uncommon at a later stage in the disease, with or without loss of consciousness, but with twitchings, and even marked convulsions. They may be frequent, or only occasional, and each leaves the patient worse than he was before. The epileptiform are more common, but the attacks are not true epilepsy or apoplexy.

The disease usually runs a slow progressive course, beginning with a definite mental change, loss of sequence of ideas, and lapses of memory, while the patient may develop sudden business energy, generally misdirected, with grandiose ideas and ambitious aims and projects, which land him too often in financial ruin. In other cases mental depression is more manifest, with suspicion of persons with whom he comes in contact.

In the last stages of the disease the patient is practically confined to bed, callous to the calls of nature, and absolutely unable to take any interest in his surroundings, and death may ensue from bedsores or from pulmonary or other complications.

Diagnosis.—The recognition of the six cardinal clinical features which are generally present will help in the diagnosis of a case of this disease. In *disseminated sclerosis* the intention tremor, nystagmus, the scanning speech, and the absence of the Argyll Robertson phenomenon are usually sufficient. By many authorities *locomotor ataxia* is held to be closely associated with general paralysis, but the differential diagnosis is easy, unless where a combination of the two diseases exists.

The **Prognosis** is bad, but life may be prolonged for two or three years. The duration is much shorter in rapid cases. With care and treatment something may be done, but not much.

The **Treatment** consists in complete bodily and mental rest; and in the relief of symptoms, and especially mental exaltation and restlessness, by the administration of hyoscine, trional, etc. Rarely does antisyphilitic treatment appear to benefit the

VII. DISEASES OF THE NERVOUS SYSTEM WITH NO CONSTANT OR WELL-RECOGNISED ORGANIC LESION

1. MYASTHENIA GRAVIS

A rare disease, resembling bulbar paralysis, but without any definite anatomical change. It consists in the rapid exhaustion of certain muscles by work and by faradic stimulation. Possibly the etiological factors are wet and over-exertion. The disease affects the muscles of the eyes, the muscles of the face, the muscles of mastication and of the neck; later all the voluntary muscles may be involved. The affected muscles do not waste, at least to begin with. The nasal speech, ptosis, and what is called the myasthenic reaction are very typical. This reaction implies the gradual loss of faradic excitability when the faradic current is applied to the affected muscles. Rest restores this excitability. About one-third of the recorded cases proved fatal, but it is a slowly advancing disease, even when incurable, and of the remaining recorded cases many seem to have entirely recovered.

The **Treatment** consists in rest and massage. Strychnine and potassium iodide should be tried.

2. PARALYSIS AGITANS

(PARKINSON'S DISEASE—SHAKING PALSY)

A chronic disease of the nervous system, characterised by a peculiar tremor associated with muscular weakness and rigidity.

Etiology.—It generally occurs between the ages of 40 and 70, and somewhat more frequently in males. Hereditary predisposition is doubtful, but there is often a family history of nervous disease. Cold, damp, anxiety, fright, an accident, and a continued fever have all been believed to be predisposing or exciting causes in different cases.

Pathological Anatomy and Pathology.—No constant pathological change is present, but minute patches of sclerosis have been described situated around the vessels of the medulla and spinal cord, while pigmentation of ganglia cells, hæmorrhages,

and obliteration of the central canal of the cord were found in certain instances. Most of these might be due to senility. Very probably the most important changes are in the nerve cells of the cerebral cortex, medulla, and cord, but whether due to a toxin or not we do not know.

Clinical Features.—The disease usually commences in an insidious manner, and the following typical phenomena are present :—

(1) *Tremor*.—This begins in one hand, with either pronator-supinator, or pill-rolling movements. As a rule the tremor is limited to the hand and wrist. In the leg the movements are best seen at the ankle-joint. Often one side is affected before the other, and the head movements, which are less common, are generally vertical, and not rotatory (as they are in senile palsy). The movements are about 5 per second, are at first under voluntary control, and are well seen when the limb in which they occur is raised, although they do not entirely cease in the attitude of repose. They stop during sleep, and are apt to be exaggerated by emotion. The handwriting is at first good, but soon voluntary control only permits of single letters being formed at a time, and even these are tremulous. In later stages of the disease the movements become much exaggerated.

(2) *Muscular Weakness and Rigidity*.—Loss of power is very apparent, and may precede the tremor, although it generally advances simultaneously with it. Associated with this is rigidity, to which the slow, stiff, voluntary movements are due, and also the tendency to flexion of many joints, as well as the typical attitude and gait of the patient.

(3) *Attitude and Gait*.—Rigidity and flexion are well seen in the attitude of the patient. His head is bent, the elbow is flexed, the face so expressionless as to be termed mask-like, and there is almost complete loss of ability to display any emotion, while the lips and tongue acquire a rigidity which renders speech slow and monotonous at first and later rapid. The intellectual sharpness is in marked contrast with the vacuous expression.

The gait is typical. He rises with difficulty, perhaps after several failures, from his chair, and with head bent forward he starts to walk with slow steps, which soon become faster and faster, as if he were running after his centre of gravity. This

is termed *propulsion*, while by the term *retropulsion* is meant the result of impelling him backwards, when, after similar rapid but retrograde steps, he often ends his career by falling. The gait is also described by the term *festinant*. In the later stages of the disease the elbows, knees, and other joints may become ankylosed in the position of flexion.

There are no alterations in superficial or tendon reflexes, or in electrical reactions.

Certain sensory changes may be present, and especially a hypersensitiveness to cold, and a tendency to profuse perspiration. In very severe cases a considerable amount of pain is not infrequently present.

Diagnosis. — *Senile tremor* has not the characteristic pronator-supinator movements, and the head is generally more markedly affected. In *disseminated sclerosis* there is exaggeration of tendon reflexes, nystagmus, and scanning speech, and the jerky movements are quite distinctive.

Prognosis. — The disease is long protracted, and not necessarily immediately serious, but cure is rare. The duration might be safely reckoned as extending for years.

Treatment. — There is no satisfactory treatment. Try arsenic, and, in severe cases, hyoscyamine or hyoscine, and bromides. Recommend a period of physical and mental rest, and give general tonics. Locally massage is often of benefit, and galvanism has occasionally been used with temporary improvement.

3. EPILEPSY

Sudden loss of consciousness, with tonic and later clonic spasms.

Etiology. — It seems probable that there are predisposing as well as exciting causes, although many of the so-called exciting causes may be rather the result of the condition than related to it etiologically. The disease generally begins before the age of 20, and in many cases at even an earlier period; certainly three-quarters of all cases of idiopathic epilepsy commence before that age. Females suffer rather more frequently than males, although in childhood there is little difference as regards sex, but towards puberty the predisposition to epilepsy is greater in the female sex.

There is a strong hereditary tendency to epilepsy, and

especially in families who are rightly called neurotic, and in whom a family history of insanity exists. Alcoholism in the parents tends to produce epilepsy in the child, and undoubtedly syphilis, although not such a potent factor as at one time it was believed to be, is still responsible for a certain proportion of cases. The so-called exciting causes are fright, which probably rarely or never produces true epilepsy; injury; fevers; some peripheral irritation, such as a tight prepuce or inflammation of the gums associated with dentition, and more rarely errors of refraction, a foreign body in the ear or nose, or the presence of worms in the rectum. These have all led to fits in young children, although such fits cannot be designated true epilepsy.

Toxic agents produce a special type of epilepsy; for example, in uræmia the seizures are epileptic in nature, and in lead poisoning, epileptic manifestations have been observed.

Vascular and cardiac conditions leading to cerebral anæmia tend also to produce epileptiform seizures.

Organic lesions of the brain and membranes, and a spicule or bony node growing from the inside of the skull, have been found in cases of epilepsy, although more commonly in the type of epilepsy termed Jacksonian.

Pathological Anatomy.—There may be organic disease, such as tumour, abscess, gumma, etc., but where the condition is idiopathic many varied microscopic lesions have been described, especially changes in cortical and other nerve cells, but none of these can be called constant. Bevan Lewis, for instance, has noted fatty changes in the nuclei, and vacuolation and disintegration of cells in the second layer of the cerebral cortex in a number of cases.

A fit seems really to be a discharge of nerve energy, and the typical aura points to the cortex as the likely centre of the nerve storm. The theory that vasomotor spasm is the cause of the fit is probably incorrect.

Clinical Features.—Many types are described; of these the most important are:—

I. **The Grand Mal.**—There is generally a definite *aura*, or warning of the approach of the fit, and this *aura* may indicate the site of disturbance and possibly even the means of treatment. The *aura* may be (a) sensory, sometimes a feeling of discomfort over the region of the stomach or abdomen, or a

tingling or a feeling of numbness in one hand, arm, or leg; or (*b*) it may be a special sense impression, such as a peculiar taste or a flash of light; while (*c*) a psychical *aura* is occasionally experienced. Good examples of this kind of *aura* are the appearance of some imaginary face, or a dreamy sensation of something morally wrong. (*d*) A motor *aura* is occasionally present, generally in the form of a spasm, associated with a certain group of muscles. The *aura* may give sufficient intimation to enable the patient to sit down or throw himself on a bed or chair so as to avoid injury.

Immediately before the fit occurs the patient emits a loud *cry* or *groan*, then suddenly loses consciousness, and falls down if he is standing, and the muscles of the body are thrown into a condition of violent *tonic spasm*. The spasm is very typical: the head is thrown back, the arms flexed, the hands clenched, the legs extended, the respiratory muscles in a state of rigid spasm, and as a result the face, at first pale, soon becomes cyanosed, and the veins of the neck and face engorged with blood. The pupils are widely dilated, and the eyes are fixed in an unnatural stare, sometimes being turned upwards. This stage lasts a few seconds, and is followed by the stage of *clonic spasm*, during which jerking movements occur generally at the rate of about three a second. All the voluntary muscles of the body may participate in the clonic movements. The eyes roll, the eyelids open and shut, the tongue is apt to be bitten by the teeth, and the patient froths at the mouth, especially during the terminal clonic movements, which are usually of special severity. This stage lasts from two to three minutes, and often in this or the preceding stage the patient evacuates the bladder or bowels. The breathing during the clonic stage is peculiarly stertorous, and this continues until the clonic movements have subsided, and the patient passes into *the stage of somnolence or sleep*, in which he remains for one, two, or more hours. Occasionally coma follows the fit, but as a rule only in cases of great severity, in which fit follows fit, and in the intervals the patient remains absolutely unconscious; this condition is termed the *status epilepticus*. In the *status epilepticus* the temperature nearly always rises, and death may occur from exhaustion. Uræmic convulsions very frequently take the form of the *status epilepticus*.

Before a fit the patient is often drowsy, and has a feeling

as if something was going to happen, while headache is not uncommon. After a fit is over, the patient is brighter, once the stage of sleep has passed off, although in certain cases, as the result of a fit, headache may persist for some time. Sometimes fits are associated with insane acts or hallucinations, and not infrequently assault and murder have been committed while in other patients the post-epileptic condition resembles hysteria more than anything else¹. It is true that the patient may remember nothing of the attack, and that peculiarities of temper are present after epileptic seizures, but unquestionably cases of post-epileptic insanity are by no means common.

The knee-jerks are frequently lost for a short time after a fit, but this loss is soon replaced by exaggeration, and in certain cases there may be local paralysis or local anaesthesia, although rather in cases where the fits are of Jacksonian nature.

It is not uncommon to find traces of sugar and albumin in the urine after a fit, but their significance is not often important.

II. Petit Mal.—All epileptic persons are subject to *petit mal*, and there are cases of epilepsy so mild that *petit mal* may alone be present. It consists in a *brief loss of consciousness*, in which frequently the patient is quite unaware that anything has happened. He may suddenly forget what he is saying when engaged in conversation; it may be, in fact, merely a sudden lapse of memory, and in a moment he resumes the interrupted conversation as if nothing had happened. Occasionally an *aura* is present with these minor fits, but as a rule there is merely the sudden unconsciousness, with pallor of face, fixity of eyes, and momentary cessation of breathing. Sometimes the bladder is emptied during a minor fit, but the tongue is never bitten, and very rarely does the patient fall down. The pupils, however, dilate even in this mild form of epilepsy, and there is often alternate dilatation and contraction for some little time after the fit is over. There are no distinct convulsive seizures, although a few tonic and clonic movements may be noted, while the lethargic state which so constantly follows the *grand mal* is not generally present.

Patients who suffer from either *grand mal* or *petit mal* very frequently begin to do so at night and nocturnal enuresis (wetting the bed) or the patient awakening with a headache.

¹ Post-epileptic automatism, as it is called—is a still more frequent, judging at the clothes, scratching, or some equally noxious performance.

in the first instance, be the only indication that something unusual has occurred. Vomiting occasionally follows an epileptic fit, and may always do so in certain patients. The number of fits vary greatly; there may be only one seizure in a number of years, or there may be several attacks, both *grand mal* and *petit mal*, in one day. Some epileptics have their fits only during the night, whilst others have them also in the day-time. Not infrequently fits occur in a longer or shorter series, and then there is an interval during which the lesser type of fits alone troubles the patient; in women the menstrual epoch is a common period of attack.

The mental condition of epileptic patients forms an important but often a distressing feature in many cases, some patients develop idiocy, others show serious and increasing mental excitement, amounting almost to mania, and in not a few cases delusional insanity is associated with the fits—in fact, many asylum inmates owe their mental condition to epilepsy. In other cases epilepsy dulls the mind, and causes considerable loss of memory, and it is only in the milder type of cases that these phenomena are unimportant and can be neglected. Possibly much of the mental deterioration is in reality the result of bromides given for too long a time and in excessive doses, but in every case of epilepsy it should be remembered that there is a tendency to the development of insanity, and that homicidal, suicidal, and imbecile types may be met with. Many of the epileptic asylum inmates become extremely dirty in their habits, indulge in masturbation, and are very irritable and remarkably destructive.

There is a characteristic type of expression termed the *epileptic facies* which is peculiar to patients suffering from epilepsy of severe type. Further, the extremities become blue and cold, there is marked constipation, and often a large appetite. Headache, mental confusion, and vertigo are also frequently complained of.

III. Sometimes fits are associated with **Insane Acts** or **Hallucinations**, and while it is necessary, from a medico-legal standpoint, to lay special stress upon this type of epilepsy, it does not require any further description. Insane acts or hallucinations occur after the fit, and for these the patient is quite irresponsible.

IV. **Jacksonian Epilepsy.**—This is the form

generally owing to a lesion of the cortex, there are localised spasms (mono-spasm), sometimes extending so as to become general. If localised, they may occur without loss of consciousness; if they become general, consciousness is nearly always lost. A localised and temporary paralysis may follow the attack, and sometimes it is associated with a localised numbness or tingling in the part affected. It should be remembered that this form of epilepsy is the type most frequently met with in uræmia and general paralysis of the insane. Jacksonian epilepsy is referred to under cerebral localisation and cerebral tumours, but it has a close connection with idiopathic epilepsy which renders it necessary to describe it here.

Diagnosis. — It is impossible to mistake an epileptic seizure which is typical, but nocturnal attacks are often overlooked. As already noted, enuresis, biting the tongue, mental confusion in the morning, and a headache, may suggest that a fit has occurred during sleep.

Faints of syncopal nature are often simulated by fits of the *petit mal* type. It is important, in connection with diagnosis, to investigate carefully the existence of an *aura*, and if convulsions occur, to get an accurate description of the attack. In *hysteria*, the patient is emotional, and the movements are excessive in extent, while the patient never bites the tongue, and does not fall down, unless a couch or bed is in a convenient position. The duration of the hysterical attack extends for a far longer time than an epileptic seizure, and it is rare to find a period of sleep or stupor associated with the attack. A hysterical patient rarely suffers from enuresis, and defæcation does not occur during the attack. It should, however, be remembered that epileptics are apt to become emotional, and that it is extremely common to find eventually a combination of hysteria and true epilepsy in the same case.

There is a form of epilepsy associated with an old hæmorrhage or patch of sclerosis or softening in the brain. In these cases the attack begins like a Jacksonian seizure, and the paralysed side is the one more affected. The attack usually occurs where the lesion is of infantile development and the mental condition of the patient generally approaches idiocy.

Prognosis. — Death almost never occurs in an epileptic

fit, but the possibility of cure depends on many things. It is distinctly more favourable when epilepsy begins after the age of 20, and when treatment is commenced before the fits have gone on for over a year. Unfavourable cases are those in which the mind has become impaired, and certainly where the family history includes epilepsy in the parents, or other serious and constitutional nervous disease. There is the risk also, in most cases, of the patient damaging him or herself during a fit, and cases are recorded where fracture of the skull has resulted from a fit occurring in the street. It is important, therefore, to remember that the occupation of the patient may expose him to great danger, and will greatly affect the prognosis as regards long life.

Treatment.—Every effort should be made to try and find out some definite cause for the attacks. Remember that there is no better method of treatment for epileptic children, where the epilepsy is severe, than to have them trained up in surroundings in which they will be free from being teased, and will not constantly have their disabilities forced upon their notice. Certain cases should therefore be brought up in asylums or in epileptic colonies, whereas others should be taught at home, if this is possible. A healthy outdoor life with little excitement, plenty but not excess of good food, and complete abstinence from alcohol of every kind, should be laid down as the best method of treating adult patients. Patients who have a definite *aura* prior to a *grand mal* do not need the constant supervision to prevent injury which other patients who have no such *aura* may require and occasionally a fit can be prevented by utilising the information obtained by the *aura*. For example, if the *aura* be a sensory impression passing up one arm, compression of the arm may prevent the fit occurring. Unfortunately, in the majority of cases such an attempt at treatment is of little avail.

The medicinal remedies most frequently given for epilepsy are the bromides. The sodium, potassium, and ammonium salts in equal parts are given 20 to 50 grains of the mixture taken three times a day after each meal. The potassium salt has the closest reputation for its effectiveness, being superior to the others. It is difficult to say whether the bromides are better than any other. Some cases seem to be benefited more by the administration of stramonium, opium, or digitalis, but the results are not so certain.

reality much better than the others. Many physicians prefer one big dose of bromide, such as a drachm at bed-time. In some cases it is given before, in other cases it is given after, food, but it must be remembered that bromide should never be persisted in for longer than 6 or 8 weeks without intermission, because there is no doubt that it dulls the intellect and affects the memory, even apart from the epilepsy, which acts in the same way. There is also a risk of bromide acne and bromism occurring when the drug has been taken for a considerable time, and especially in patients who have any idiosyncrasy. Sometimes the use of arsenic along with the bromide prevents this trouble; in other cases the drug should be stopped for some weeks.

Cod-liver oil and iron are probably as important as bromides, but after treatment with bromides has resulted in amelioration or cure, the case should be kept under observation, and bromides in lessened doses given from time to time; in women, for example, they should be given at the menstrual period.

Belladonna, chloral, and cannabis indica form a group of remedies of considerable value, and they may take the place of the bromides when these drugs have been intermitted. Amongst other remedies suggested are ergot, antipyrine, nitroglycerine (with the idea that a fit is the result of vasomotor spasm), zinc and silver salts, borax, and many other drugs, but it is difficult to recommend one of these more than another. Opium has also been given, but its administration should be kept under very strict supervision.

No case of epilepsy in children should escape the most careful and painstaking search for some exciting cause; a tight prepucce, the presence of worms, any irritation in nose or ears, in short anything likely to act as a peripheral irritant, should claim the attention of the physician.

The surgical treatment of epilepsy is not very hopeful: sometimes the *aura* suggests a site of irritation which can be localised in the brain, or tapping with the finger on the patient's head may elicit pain over one part of the bone, and under these circumstances trephining is warranted. In Jacksonian epilepsy it is needless to say that operative interference is often of the greatest service.

Epileptic colonies have been for long established on the

Continent, and there are already several in England, such as those at Chalfont St. Peter, Buckinghamshire, and Maghull Epileptic Colony near Liverpool, and the system of finding suitable work for epileptics and having them congregated together in one institution has proved of great benefit.

It should be stated that under no circumstances is the marriage of epileptic persons to be encouraged, because epileptic children are an extremely common result of the union of epileptic parents, and even where only one parent is so affected.

ECLAMPSIA

This is closely allied to epilepsy. Eclamptic seizures are more common in children, but they differ from true epilepsy inasmuch as there is no tendency to many attacks. It generally occurs in children up to the age of two years, it is rare after five, and exceptional after seven.

Etiology.—The *predisposing* cause is unquestionably the unstable condition of the central nervous system in early childhood, an instability specially marked if the child be weakly, subject to rickets, or the offspring of highly neurotic parents. Anything causing debility, and many continued fevers, such as scarlet fever, measles, pneumonia, and whooping-cough, may be associated with one or more eclamptic seizures, and in the continued fevers in children a fit is often the initial manifestation.

The *exciting* causes can sometimes be differentiated from the predisposing. The irritation of the first dentition, some gastric or intestinal disturbance, and the presence of worms, may cause fits; while a tight prepucial band, inflammation of the ear or nose, a foreign body in the external auditory meatus, and even a full bladder, may be sufficient to produce one or more fits in children who are predisposed.

Clinical Features.—Without any warning the child begins to show twitching movements often associated with grinding of the teeth. There may be a definite tonic spasm during which the body becomes rigid, the breathing ceases, and the face, after being pale, becomes blue and cyanosed. Clonic spasms follow, the eyes roll, the mouth and face muscles twitch, and twitching movements may be noted in arms and legs. It is common to find in children what has been described as carpo-

pedal spasms, by which is implied the bending inwards of the thumbs and the great toes; but such spasms, though they may occur in connection with the fits, are often distinct from true childish eclampsia. There may never be more than one fit, or there may be several, which, however, do not recur when the peripheral irritant has been removed.

The **Treatment** should consist in giving an emetic or purge where the alimentary tract is at fault, in examining and possibly lancing the gums, and in removing any foreign body from ear or nose, should such a foreign body be present. A hot bath is often most important, and a warm sponge placed over the chest gives relief in cases in which the fits recur frequently, while the administration of chloroform to produce general anæsthesia is sometimes called for. Occasionally anti-spasmodics require to be ordered, and especially bromides or chloral, which should be administered in small doses. The child's general health should receive careful attention and tonics, and good food must be given.

Eclamptic seizures also occur where there is meningitis or inflammation of the brain, but these attacks are not the same as the reflex eclamptic seizures to which we have just referred.

4. CHOREA

(ST. VITUS'S DANCE—SYDENHAM'S CHOREA)

A disease associated with, as yet, no definite organic lesion, but characterised by involuntary, inco-ordinate, and jerky movements of one or more limbs and often of the facial muscles, and with a close relationship both to rheumatism and to endocarditis.

Etiology.—Chorea may be the result of the following conditions :—

(1) Some peripheral irritant which, however, must be co-existent with a neurotic disposition, or at all events with what is generally termed a "highly-strung" nervous system. Examples of such peripheral irritants are a long prepuce, the presence of thread-worms, and occasionally the irritation associated with dentition. These causes are more common in the child, whereas in the adult, pregnancy is sometimes respon-

sible for one of the most severe types of chorea, in which the severity of the attack may even endanger the life of the patient.

(2) Many cases of chorea are directly related to rheumatism. There is a family history of rheumatism and generally a previous record of muscular or joint pains, while at the time of attack, rheumatic manifestations may be replaced by the choreic movements.

(3) It is difficult to classify endocarditis as an etiological factor distinct from the preceding group. It is probable that it is in patients who suffer from rheumatism and also from endocarditis that chorea is most common, and it has been thought that small portions of clot from vegetations may, as emboli, represent the causal organic lesions in the neighbourhood of the basal ganglia or of some other important centres in the nervous system.

(4) There is no question that a fright or a severe scolding may be sufficient to start chorea in a neurotic subject, and we may include under this group the chorea so frequently met with in children who have been overworked at school. There are also cases in which imitation of a comrade affected with the disease is mainly responsible for the attack.

Pathological Anatomy.—Much discussion has arisen with regard to the site of the supposed organic lesions, and whether they should be sought for in the cortical cerebral convolutions, in the region of the basal ganglia, and perhaps specially the optic thalamus, or in the groups of motor nerve cells in the anterior horn of the cord. The theory that multiple emboli, due to endocarditis with vegetations, account for the majority of cases, is not supported by the results of pathological research. It is true that the introduction of fine particles of sand into the carotid blood-stream of the dog causes choreic movements, but in cases which have proved fatal in the human subject there is often no evidence of endocarditis with vegetations, and also no small patches of softening such as would be caused by the smallest emboli. Recently an infective organism, a diplococcus, has been described by Messrs. Poynton and Payne, in connection with the joints and cardiac lesions in acute rheumatism, and chorea has been produced in animals in, at all events, certain cases in which this organism was injected. It seems, therefore, possible that there may be some truth in the embolic theory, although our information as to the exact nature

of the changes present is, as yet, very inadequate. The old-fashioned opinion that instability of important brain centres explains chorea most satisfactorily, has still many supporters, and has much to be urged in its favour.

Clinical Features.—The characteristic features of the movements in chorea are:—

(1) They are involuntary; they are increased by nervousness and by any voluntary effort.

(2) They are jerky, the jerkiness being of an inco-ordinate type, and they absolutely lack rhythm.

(3) They are generally unilateral, so that a hemichorea is the most common form, although the opposite arm and leg may be more slightly affected.

As regards the face movements, they may be described as jerky grimaces with winking of the eyes, and twitching movements of the corners of the mouth, more marked on the affected side; and if the patient is directed to put out the tongue, it is often jerked out and as quickly drawn in, while in certain cases the lower jaw has involuntarily closed on the tongue, biting it severely. The muscular weakness associated with the choreic movements may actually amount to a definite paralysis. As a general rule the patient is able to swallow freely, and respirations, although slightly jerky and irregular, show no marked alteration otherwise. Sometimes deep sighing inspirations are noted, and in severe cases the contraction of the diaphragm may not be quite rhythmic with the movements of the intercostal muscles.

In very severe cases the movements of the limbs are violent, and if the patient is not protected by pillows, serious abrasions may be caused, and indeed in exceptional cases the movements are so violent as to necessitate the use of a mattress on the floor, so as to obviate the possibility of the patient tumbling out of bed. In such patients, speech is generally considerably involved, and not infrequently, in the more neurotic type of children affected with this disease, there may be great peculiarity of temper with much display of emotion, difficulty in speech, and sometimes even difficulty in swallowing.

The inevitable result of a prolonged attack of chorea is the exhaustion of the patient, and when there is sufficient restlessness to prevent sleep, and a tendency to maniacal manifestations, the case is always grave. Generally there are no sensory

changes at all and the reflexes are unaffected or slightly exaggerated. Very rarely the affected limbs are tender to touch. The temperature, excepting in cases of great severity and especially where the disease is associated with pregnancy, is usually normal, and generally pyrexia only accompanies maniacal excitement, where it is present.

The cardiac condition should invariably be investigated. It is common to find hæmic murmurs, especially where the patient is bloodless, but mitral stenosis is also common, indicating the presence of endocarditis not necessarily of a recent nature. Irregularity of the cardiac rhythm has been described in chorea, and it has been suggested that it is due to choreic contractions of the papillary muscles of the heart, and that this explanation may account also for the murmurs so often present; but it may be pointed out that it is the voluntary muscles alone which usually suffer, and therefore this explanation is untenable, and the cardiac irregularity is more probably the result of nervous exhaustion than of anything else.

Attacks of chorea usually subside in three to six weeks, but a relapse is not uncommon, and the causal factors in each case have much to do with the probable duration.

Diagnosis.—It is easy to recognise chorea by making the patient either extend the fingers and attempt to keep them at rest, to put buttons into buttonholes, or to tie knots on pieces of string. The latter movements demand accurate effort and perfect co-ordination, and the attempt to perform them brings into prominence the jerking movements peculiar to the disease.

Care must be taken not to confuse *athetosis* with chorea. Athetoid movements are slow, rhythmic, involuntary movements confined usually to one hand, possibly to one hand and foot, generally associated with infantile hemiplegia or some cerebral lesion and often accompanied by mental debility, while the fact that the affected limb or limbs show a more or less distinct paralysis generally prevents any difficulty in the diagnosis, because paralysis is rare in chorea.

Prognosis.—The only serious cases are those in which the movements are very violent, and these are not infrequently associated with pregnancy, but the cardiac condition may give rise to anxiety, especially if endocarditis is present and is acute and ulcerative. Cases with temperature, and therefore with more or less delirium, are less favourable, but a great deal

depends upon skilful nursing, so that a sufficient amount of food and sleep may be obtained by the patient.

Treatment.—The stock treatment for chorea is arsenic, and probably there is no remedy of such value, and which can be given for almost any type of the disease, the dose varying with the age of the patient, but generally beginning with 3-5 minims three times a day. The peripheral irritant, if there is one present, should always be looked for and removed, and attention directed to ensuring as much rest as possible, and the prevention of damage to the patient, should the movements be violent. Generally in these cases a mattress placed on the floor and surrounded with pillows is better than an attempt to restrain the patient in bed, while a good deal may be done to limit the movements by keeping the patients absolutely quiet, free from excitement, and in certain cases isolated. Food should be given in easily digested and easily administered forms, and fish cream, pounded meat, mince, and beef-tea, and of course milk, are all admirable, although it should be remembered that fluids may be swallowed with greater difficulty than semi-solids.

As regards drugs, with the exception of arsenic it is difficult to know what to recommend. Certainly in rheumatic cases salicylate of soda is of great value, and bromides sometimes alone, sometimes associated with chloral, have proved efficacious. Antipyrine in moderate doses has acquired a considerable reputation, but it should be used with care. Remember that although arsenic is probably the best remedy, it should never be given unless under strict supervision, because arsenical poisoning, if not arsenical neuritis, may be produced, and may cause much and prolonged suffering. The dose, therefore, while increased gradually, should, in most cases, never exceed 10 minims of Fowler's solution three times in the day.

Where there is intense delirium, cold sponging and the use of the ice-bag are advantageous, and to ensure a certain amount of rest chloroform has been administered as a general anæsthetic.

CONDITIONS ALLIED TO CHOREA

I. CHRONIC CHOREA, HUNTINGDON'S CHOREA

There is a form of chronic chorea which is frequently called Huntingdon's chorea, and which is characterised by

irregular, choreic-like movements with difficulty in speech, gradual and progressive dementia, and a strong hereditary tendency in not a few cases. It begins at or after middle life, affects both sexes, and occurs in the members of a particular family. Huntington, who first described the disease, has records of a case in which preceding generations had been treated by his father and grandfather, who were physicians before him. The speech is peculiarly slurring and the gait unsteady, but the movements described as choreic are perhaps rather inco-ordinate, while the progressive failure of the mental powers is a typical feature of all cases. The patients rapidly become insane, although the insanity rarely assumes, even for a time, an acute stage. Little is known of the pathological anatomy of the disease, owing to its excessive rarity, but atrophy of the cerebral convolutions, and in other cases scattered patches of sclerosis have been described. It is hardly necessary to state that the prognosis is bad, and that the treatment is most unsatisfactory.

II. SENILE CHOREA

Occasionally in old persons choreic movements develop as life advances. There is no hereditary tendency and no mental weakness, but the movements, once started, generally continue and probably annoy those persons who come in contact with the patient more than the patients themselves.

III. CHOREA MAJOR, OR DANCING MANIA, OR EPIDEMIC CHOREA

This is the original St. Vitus's dance, a disease, if it can honestly be so called, which dates back to the Middle Ages. It was associated with periods of excessive mental excitement, and occurred in pilgrims attending the chapel of St. Vitus in the Rhine provinces. Dancing, jumping, and gesticulations of all kinds were associated with great religious excitement, and the disease was in reality a type of hysteria.

IV. SALTATORY SPASM

This is a peculiar hysterical manifestation to which, however, a special name has been given. It is very similar to the

so-called chorea major, only that the individuals affected jump or spring in the most remarkable fashion. Often associated with this jumping movement is a tendency to explosive utterances or to the repetition of some single word.

V. HABIT CHOREA OR HABIT SPASM, CONVULSIVE TIC, AND HEAD-NODDING, ETC.

A group of affections, some of which are associated with mental disturbance, while others are not; habit spasm at all events is probably rarely accompanied by much, if any, psychical phenomena.

(a) *Habit Spasm or Habit Chorea (Convulsive Tic)*.—This is extremely common in nervous girls and children. It consists in certain movements of the head or face muscles, such as winking, nodding, twitching of the corners of the mouth, shaking movements of the head, movements of the ears and scalp, and similar peculiarities which become a habit to the individual. Many of these are learned in odd ways, and once acquired they are very difficult to get rid of. They are apt to be more marked when the individual is fatigued or nervous, and in the case of winking and other facial contortions may become a matter of great disquietude to the patient and to his or her friends.

Similar movements of the body as well as of the head may become a habit in certain persons, and these jerking movements are generally met with in neurotic individuals, and are increased by nervousness and by fatigue.

(b) *Head-nodding and Eclampsia Nutans*.—Head-nodding is an affection closely allied to habit spasm, in which there are jerking movements of the head, and in the case of eclampsia nutans bowing movements of the whole body. These movements are most common in delicate children, and rickets appears to be in certain cases associated with it. Nystagmus is not infrequently present.

Nodding and bowing movements are not uncommon in certain types of congenital idiots, while in Friedreich's ataxia there are movements which are to a certain extent slow and nodding in character.

(c) *Impulsive Tic*.—There is a group of cases in which, as the result of a peculiar psychical affection, the patient not

merely suffers from involuntary muscular movements of face and limbs, but in addition may give forth explosive sounds, sometimes in the form of cry, bark, or shout, or else the sudden and explosive utterance of an oath or some obscene word. Besides these movements and explosive utterances there is also a peculiar mental phase generally taking the form of delusions, and to this group of clinical features Gilles de la Tourette has given the name of "Impulsive Tic."

Treatment of Foregoing Conditions. — It is hardly necessary to say much about the treatment of most of the affections just mentioned; some of them certainly are incurable, but in cases of habit spasm a great deal may be done in the way of checking the peculiar jerking movements which annoy the patient. No remedy acts as a specific, although the strength of the patient should be fully maintained and tonics of all sorts are necessary. Endeavour to educate the patient to give up the habit spasm; often this requires patient and protracted treatment.

5. OCCUPATION NEUROSES

(WRITER'S CRAMP, TELEGRAPHIST'S CRAMP, PIANIST'S CRAMP)

A group of nervous affections incident to various trades and occupations, and characterised by interference with certain muscular movements which form a large part of the professional work of the patient.

Etiology.—It is due to the excessive use of the muscles which become affected, sometimes owing to improper use, perhaps in certain cases merely as the result of great stimulation of one or more groups of cells.

Pathology.—There is rarely any obvious change in the cells of the nervous system governing the affected muscles, but the following varieties of the affection, some of which imply definite pathological change, at least in the muscles, may be met with:—

(1) The condition may be a failure of co-ordinating power for a particular action constantly required in the daily work of the patient.

(2) A disturbance of, rather than a failure in, co-ordination, associated with excessive irritability of groups of muscles

which are not required for the carrying out of the action in question, but which happen to be near them anatomically.

(3) An association of the first or second type with wasting or paralysis of the muscles affected.

(4) Cramps or spasms, excessive tremor, and not infrequently pain may be associated with the affected group of muscles to a greater or less extent, and sometimes the spasm involves muscles other than those required for the movements in question, so that in writer's cramp, one of the most common types of the disease, the pen or pencil may be stuck through the paper or thrown out of the hand.

The **Clinical Features** are stated in what has just been said. In writer's cramp there is often spasm with pain, and very generally tremor involving the muscles which may in time waste. The patient may bring into play other muscles so as to get over the difficulty, but these muscles also soon become affected. There is sometimes, although rarely, a little anæsthesia, and the skin occasionally shows definite trophic changes, the fingers becoming glossy, while sometimes vasomotor phenomena develop, the patient becoming subject to chilblains. Electrical irritability is often diminished in the affected muscles.

While this description specially refers to writer's cramp, similar trade spasms are met with in violinists, pianists, hammermen, and telegraphists, especially those who work the Morse instrument, in the use of which Gowers estimates that over 50,000 similar movements were made by the patient in one day. Closely allied to these trade spasms is miner's nystagmus.

The **Diagnosis** of writer's cramp or palsy is easy. There is nothing with which one can confuse it, but, on the other hand, nervous people are apt to imagine that they are the victims of the affection.

The **Prognosis** is unfavourable, because even if the left hand be used for writing, or the patient learn to write from the shoulder, it is by no means certain that the disease will not attack the newly educated muscles.

Treatment. — Certainly it is desirable to interdict all writing, and it might be added all occupation causing other forms of trade spasm. Rhythmic exercises together with galvanism have been recommended, and attention should be paid to the patient's general health.

6. TETANY

Tonic spasms, generally limited and chiefly affecting the extremities.

Etiology.—There are several distinct varieties of tetany to which reference must be made.

It may be present in children in connection with severe diarrhoea, and in adults who are debilitated in consequence of prolonged lactation or after any continued fever. It has been met with in cases of dilated stomach, and a form of tetany is due to extirpation of the thyroid and parathyroid glands together. Lastly, epidemics of tetany have been described, the exact nature of which seems somewhat doubtful. Such epidemics occurred in Paris in 1855 and 1876, and they were more common during the winter months.

Clinical Features.—In cases associated with debility there are what have already been described as carpo-pedal spasms, in which the fingers are closed on the thumb, the terminal phalangeal joints being extended, the feet are extended, and the toes adducted. Sometimes the muscles of the face and neck are involved, and there may be trismus. These attacks occur in paroxysms, and what is described as a paroxysm may be produced by pressure on the affected part, either on the nerves or on the chief blood-vessels, while tapping the affected muscles tends to throw them into active contraction. The electric reactions are often greatly increased, and there may be a certain amount of pain associated with the condition. The attacks last for some hours.

Diagnosis.—It is easy to recognise the carpo-pedal spasm so common in children, but many authorities hold that it is hardly analogous to tetany. It is impossible to confuse tetany with *tetanus*, and it is rare to find a *hysterical* imitation close enough to render a mistake possible.

The **Prognosis** is favourable, and the **Treatment** consists in the use of hot and cold baths alternately, sometimes the administration of bromides, and in very severe cases chloroform anæsthesia. The ice-bag, general massage, and the various forms of electricity have often proved of value; in cases where the thyroid has been extirpated, thyroid extract should invariably be administered.

7. PARAMYOCLONUS MULTIPLEX

A remarkable disease, in which clonic contractions of certain muscles of the limbs occur, the contractions being sometimes constant. These contractions are apparently the result of fright or great emotional excitement, and in certain cases the most violent movements are met with. There is occasionally a hereditary element, but the disease is rare and may be associated with other affections of the nervous system.

8. MIGRAINE OR HEMICRANIA

Paroxysmal attacks of headache, generally unilateral, with sickness, vomiting, and often peculiar disturbances of vision.

Etiology.—The *predisposing* causes are hereditary, and especially in the neurotic or gouty. Commoner in women, the headaches may begin in childhood, and continue throughout life, and they are more frequent in winter. The *exciting* causes are: peripheral irritation from nose, eyes, teeth, or pelvis; fatigue; mental emotions; overwork; gastric or intestinal disturbance.

The **Pathology** is still doubtful. Neuralgia of the 1st division of the 5th nerve, vasomotor spasm followed by dilatation, and a "nerve storm" in a sensory centre are all theoretical explanations which have been suggested.

Clinical Features.—Often after premonitory symptoms of malaise, depression, etc., a severe pain begins in one temple or other part of the head, rapidly increasing in intensity, generally with nausea, and in a large number of cases with dimness of vision, flashes of light, and occasional hemianopsia. The attack lasts for some hours or a day, and frequently passes off with vomiting. The attacks may be definitely periodic.

Prognosis.—It tends in many cases to recur, but does not endanger life.

Treatment.—Remove any exciting cause and study the constitution of the patient. For an attack order rest in bed, promote vomiting in gastric cases by ipecachuana or other means, and then administer antipyrine, phenacetin, salicylate of soda, or similar remedy. Opium and the inhalation of chloroform often relieve, but are dangerous remedies. On the assumption of the correctness of the vasomotor theory, nitroglycerine has been suggested and found useful in some instances.

9. NEURASTHENIA

Loss of nerve energy, together with irritability of many nerve centres.

Etiology.—The *predisposing* causes are hereditary influence, unwholesome life as regards the mode of upbringing and education, excessive excitement, and mental overstrain. Certain races, as for example the Jews, appear to be specially predisposed.

The *exciting* causes are overwork, and particularly mental overwork, emotional excitement and nervous exhaustion, fevers, any severe illness, and dissipation. Perhaps the most marked cases of neurasthenia are met with in persons addicted to the drug habit, and especially morphia and cocaine. A mental shock, such as a severe accident, is more likely to leave its mark upon patients predisposed to neurasthenia than on healthy strong-minded persons, but even in the latter it may start a definite neurasthenia.

Clinical Features.—All the manifestations which one anticipates meeting with in general debility may be present, and in addition there is pain in the back, a feeling of oppression on the top of the head, vague sense of discomfort, often palpitation and pulsating abdominal aorta, headache, and very generally disorders of the mind and memory and sometimes of the special senses, while sleeplessness is peculiarly common. Some of the manifestations just referred to might be termed instances of hysteria or hypochondriasis. Considerable emotional excitement is not infrequently present, while a number of suicidal cases are in reality neurasthenics who have lost hope and become excessively depressed. Many persons suffering from neurasthenia are retiring in disposition, and dread society, and not a few of them imagine they suffer from some serious disease. The peculiarities of vision include tiredness after any short effort of sight, and there may be a similar peculiarity with regard to hearing. Many of the typical phenomena are associated with the spine, and these include the pains already described, which may be peculiarly severe in patients who have the spinal type of neurasthenia, a type very common after a railway accident. Sometimes paralyses of different kinds develop, and not infrequently there are peculiar vasomotor phenomena such as general or localised sweating,

over the chest, throbbing of vessels, and other visceral sensations. A painful corresponding ovarian tenderness in some cases.

~~Requires~~ requires the same care as hypochondriasis. It is necessary to recognise the physical debility of the patient, and sometimes possible to exclude any organic cause. The symptoms complained of may have suggested the diagnosis.

~~Prognosis~~ is often favourable and depends largely on the patient's effort to improve the condition.

~~Various~~ Remedies of all kinds should be administered, when the physical strength has been sufficiently restored. Fresh air and scene are most beneficial, and in some cases Mitchell treatment is indicated.

Medication is of great value, and any cause for the condition should be sought for and, if present, vigorously treated. It is specially necessary in cases where the patient has the drug habit.

10. HYPOCHONDRIASIS

in which the sufferer imagines illnesses, and converts a trivial complaint into a most serious disease, and regards the state of his health as demanding the most urgent treatment.

~~Causes~~ — A neurotic family history, and mental depression or other worries, together with gout and rheumatism probably predispose to this affliction. Many of the cases are certainly hysterical, some of them suffer from moral obliquity, and many cases are to be met with in asylum inmates.

Clinical Features.—In young persons hypochondriasis is usually associated with sexual irritability or masturbation, and in other cases imaginary impotence is an important feature.

In middle-aged persons the illness imagined may take various forms. Some patients really suffer from illness, and they add to the true symptoms a large variety of imaginary ones.

imaginary affections which may render their lives almost unbearable. Pains in the back and legs, in the head, and in almost any part of the body, are extremely common. A number of these patients suffer from sluggish action of the liver associated with dyspepsia, and they may feel supposititious internal parasites, have definite evidence in their opinion of the existence of cancer, and so forth. In older patients impotence and the imaginary ill-results of indulgence in vice during early years may render much aid to the development of hypochondriasis. Syphilophobia is also a common manifestation.

The **Diagnosis** demands considerable care in order to prevent a real affection being called an imaginary one.

The **Prognosis** is often unfavourable; the symptoms imagined by the patient may persist for many years, sometimes complete recovery occurs, while in the worst type of cases the patient may commit suicide or may become insane.

Treatment.—Order change of air and scene in every case where such treatment is possible. Endeavour to divert the patient's attention from the affected organ or organs of the body, and abstain from informing him that his symptoms are purely imaginary. The first step towards successful treatment is to obtain the confidence of the patient.

11. HYSTERIA

A functional affection depending on instability of the nervous system, but which may depend to a certain extent on organic disease. There may be sensory, motor, vasomotor, psychic, and other manifestations.

Etiology.—In many cases faulty upbringing and education have much to do with hysteria. A child, weakly in body and who has never been controlled, may develop, towards puberty, the mental instability which causes this disease. Mental shock, debility from long-continued ill-health, the effects of alcoholism, syphilis, or tuberculosis, nervous diseases of many kinds, including epilepsy and insanity in the paternal or maternal history, are peculiarly apt to lead to an ill-balanced mental equilibrium in the children. Intermarriage is, very probably, another important factor in many cases.

Over-education, faulty methods of education, excessive excitement of any kind, precocious interest in sexual mat-

indulgence in masturbation, and especially during early years, are all apt to give rise to hysteria.

Granted that the patient is a suitable subject hereditarily and personally for the development of the disease, it is easy to understand how some unfortunate love-affair, mental strain, anxiety, fright, fear of an examination, etc., may upset the patient's mental balance.

Pathological Anatomy.—Unfortunately we have little to say with regard to the pathology of hysteria. It is extremely probable that there are minute, but certainly as yet unrecognised, changes in the cells of the higher centres of the nervous system.

Clinical Features.—Many pages might be written without exhausting the innumerable features met with in hysteria. It is perhaps better, therefore, to describe a hysterical seizure, and then to refer under suitable headings to the most typical phenomena which are present in different cases.

The *paroxysm* generally begins with premonitory nervous phenomena, such as depression or excitement, and then a more or less typical hystero-epileptic seizure occurs. There are tonic and clonic spasms, with often extremely marked opisthotonos, but the clonic movements are very extensive and the patient—often a female—hits out at any one near at hand, while, though falling down, she generally selects a suitable spot for the seizure, where a sofa or bed is available. After the spasm stage has passed off, there is sometimes a stage of contortions and cataleptic poses, followed by a stage during which the patient demonstrates great emotional excitement, the face being expressive of intense love, pain, desire, hate, or other emotion. Lastly, a stage of delirium or hallucinations may develop. There are areas called hystero-genetic on the front and back of the body, the most important of which are shown in the diagrams. Pressure over certain of these areas may bring on an attack, or arrest it, if it has begun. It should be noted that only certain of these areas are active in any given case. Sometimes somnambulism, catalepsy, and trance form a definite part of the attack. The paroxysms occur when the patient is surrounded by friends or has a suitable audience, and they last for a long time, especially when much sympathy is shown towards the sufferer. In many cases the patient can prevent an attack occurring, but once it has commenced it appears to be beyond the control of the patient's will-power.

It is between these paroxysms that the many sensory, motor, visceral, and psychical symptoms occur, and there are cases in which no typical hystero-epileptic paroxysms are present at all.

Before describing some of the chief phenomena associated with hysteria it is desirable to state again that the patient invariably demands sympathy, and that without sympathy many of the most typical symptoms do not appear.

1. *Sensory Functions*.—Not infrequently the patient develops anæsthesia over one-half of the body or over some part of it such as the soft palate, and similarly, areas of

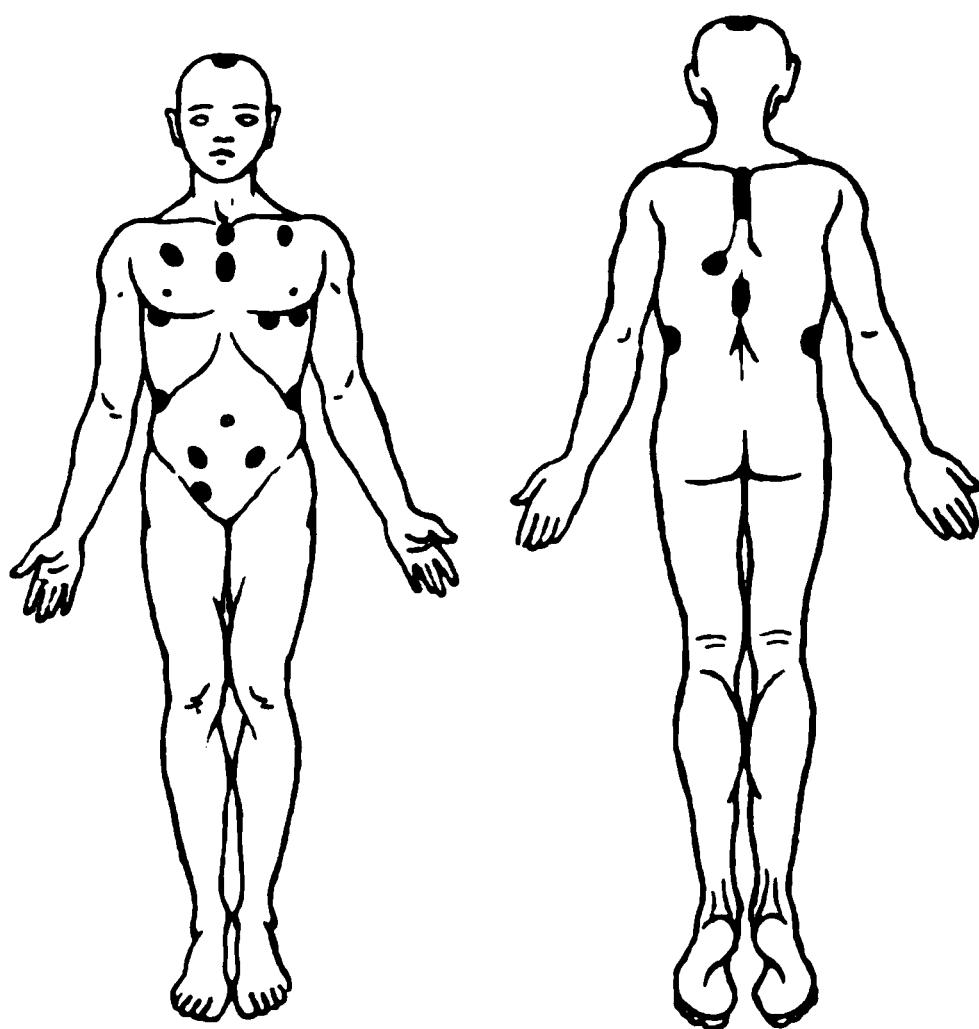


DIAGRAM 48. — Diagrams of chief Hystero-genetic Areas on anterior and posterior aspects of the body. (After Charcot.)

hyperæsthesia may also be present. It should be noted that the hystero-genetic areas already referred to are areas of hyperæsthesia. In addition it occasionally happens that certain joints become painful, the condition simulating inflammation or disease of these joints. It has been found possible, by means of magnets and other devices, to move the anæsthetic and hyperæsthetic areas from one side of the body to the other. Pains in the back, head, and indeed all over the body are not infrequent, and may be described by the sufferer as being of intense severity.

2. *Special Senses*.—Sight is not infrequently affected; the patient may be completely although temporarily blind, or may

have various forms of scotoma, while colour-vision may be greatly limited. Sometimes the conjunctiva is peculiarly insensitive. The other special senses, hearing, taste, or smell, may also suffer.

3. *Motor Functions*.—All kinds of paralysis occur in hysteria, sometimes of a limb, sometimes merely of a group of muscles, while in other cases a hemiplegia is simulated with great exactness. The paralysed muscles do not waste, and the reaction of degeneration is not obtained where in cases of an organic nature these phenomena would be expected.

Aphonia or loss of the voice is perhaps one of the commonest forms of motor paralysis, and it is remarkable how, in hysterical cases, recovery may follow some trivial procedure on the part of the physician, which could not possibly have any remedial effect whatsoever. Contractions and spasms are frequent, a limb may be flexed so as to be rendered almost useless, while the nails of the fingers may be firmly pressed into the palm of the hand. One of the most remarkable forms of spasm is that producing the so-called phantom tumour, found in the abdomen, and resembling a neoplasm in appearance. Tremors of different degrees of coarseness are also common; the movements may be fast or slow, and may be kept up in a most convincing manner by the patient.

4. *Visceral Phenomena*.—In this group there are many common hysterical manifestations. In the *Alimentary System* one finds typical spasm of the œsophagus, and the *globus hystericus* of which so many patients complain as passing up the gullet is a striking instance of this spasm. In not a few cases the patient describes the appetite as absolutely lost, while eating privately the food necessary to support life.¹ Phantom tumours have already been described, and it is probable that the remarkable rumblings, noisy eructations due to the retention of air behind the larynx or to swallowing it into the stomach, are more or less of hysterical origin. It should not be forgotten that nervousness may bring on diarrhœa, and therefore considerable alimentary disturbance may be associated with hysteria. A remarkable phenomenon which may be noted here is excessive salivation, the patient sometimes secreting and expectorating an enormous amount of saliva.

¹ *Anorexia nervosa* is the term applied to absolute loss of appetite which may continue for long periods of time.

In the *Circulatory System* palpitation is very common and may be accompanied by great discomfort and often cardiac pain.

In the *Respiratory System* the hysterical cough is one of the best recognised hysterical phenomena. It is loud, barking, and much more annoying to those around than to the patient herself. Attacks of dyspnœa may sometimes develop, the rapidity of breathing being remarkable and often most distressing. The attack wears off after a short time, and is induced by excitement or any similar cause.

In the *Integumentary System* there may be urticaria or flushing of the skin either localised or very general over the trunk. In some remarkable cases stigmata or purpuric hæmorrhages have appeared in the skin as the result of a hysterical attack. In very severe cases trophic changes may occur, and sometimes even the nails are shed.

In the *Genito-Urinary System* sexual irritability is not uncommon, while indecency and a love of obscene language and literature are occasionally present. The bladder often gives much trouble, the patient complaining of being totally unable to pass water, while in other cases polyuria is present after hysterical seizures.

5. *Mental Changes*.—The intense love of sympathy already noted dominates the phenomena belonging to this group of symptoms. The patients are generally either excited or depressed; they will tell any number of untruths provided the sympathy desired is shown to them. They are childlike in their outbursts of temper and readily become passionate. The effect of alcohol is to produce a condition almost approaching mania, and the hallucinations associated with the attacks of hysteria may take many varied forms. The patient commonly manufactures ailments so as to attract the attention of those surrounding her, and not infrequently sugar or white of egg has been added to urine, or the patient has cut herself, introduced foreign bodies of different kinds into the nose, ear, vagina, etc., and in short has done everything to obtain the medical or other interest which her mental condition demands. In a separate but very common group of cases there is hypochondriasis in which the patients imagine that they have all sorts of troubles or diseases, such as that a serpent lives inside them, that they are continually annoyed by evil spirits, and, in the more sensible cases, that they suffer from

cancer, heart disease, lung disease, or some other serious and alarming affection.

The emotional excitement, the stage of contortions and the stage of hallucinations so commonly associated with a hystero-epileptic seizure are all examples of mental phenomena. In the intervals between the attacks many psychical symptoms may develop, as, for example, *astasia* and *abasia*,¹ while the border-line between insanity and hysteria is often a very narrow one. Many patients develop hysterical manifestations in connection with religious excitement, and, just as in insanity, it is remarkable how a mixture of religious and sexual emotions may be present in the same case.

The temperature may become remarkably high, and apparent hyperpyrexia is a common manifestation of severe attacks, the patient in a mysterious way managing to raise the temperature to 110°, 115° F., or even higher, although in most cases the hyperpyrexia is due to deliberate fraud.

It should be remembered that in patients suffering from true organic disease, and whose mental equilibrium has suffered from protracted ill-health, hysteria is not infrequently associated with the clinical features characteristic of the disease in question.

Diagnosis.—As a general rule the patients are young females and possess the definite hystero-genetic areas already described. In the more severe cases *anæsthesia* of the posterior pharyngeal wall and other typical hysterical manifestations point to a definite diagnosis. A careful search must, however, be made for any organic lesion. Hysterical contractures and *pâralyses* may often be proved to be functional by the administration of chloroform, and hysterical fits are much more lengthy than true epileptic seizures.

Prognosis.—Much depends on the zeal of the physician and the patient's obtaining good and efficient nursing. It is impossible to effect a rapid cure in most cases, but no case of hysteria, however severe, should be looked upon as hopeless.

Treatment.—The Weir Mitchell method of treatment is probably the best. It consists in the isolation of the patient, depriving her of the sympathy which does so much to keep up the disease. At the same time she is kindly treated,

¹ *Astasia* is inability to stand, *abasia* inability to walk, and yet with unaffected sensation, motor power, and co-ordination.

is well fed, in fact is made to eat an excess of easily digested food, and is under the care of a strong-minded nurse or attendant. Massage is generally given and may be kept up daily for an amount of time suitable to the patient's strength. Exercise is distinctly desirable where it can be taken, and electricity in all its forms, especially static electricity and faradism, have been found of great benefit. It is useless to contradict the patient by stating that symptoms complained of are purely imaginary. Wise mental guidance and the cultivation of self-control and will-power are the proper lines of treatment.

The drugs generally administered consist of valerian, of which 30 minims of the ammoniated tincture may be ordered, or musk, and the ordinary tonics, but probably it is not so much the drug as the objectionable taste of certain remedies such as valerian and assafœtida which is responsible for the benefit obtained from their administration.

Unquestionably hypnotism is a powerful agent, but its use rather weakens than strengthens the patient's will-power and self-restraint. While, therefore, it may be prudent to treat the patient by suggestion, it is certainly foolish to resort to hypnotism in its ordinary form.

For hystero-epileptic fits the order to place the patient in a cold bath, should a subsequent fit be taken, or to pour cold water from a height on to her face, will often arrest a sequence of seizures, and the more uncomfortable the treatment the better it operates.

PROPHYLACTIC TREATMENT.—Parents should endeavour to enforce restraint on children, and particularly in the case of those who are delicate, physically or mentally. Self-control is probably better taught by parents and nurses during the early years of life than by any other means later on. Care should also be taken to prevent children allowing their minds to dwell too much upon one subject, however right and proper that subject may be, and variety of mental work with a sufficiency of physical exercise should be the régime for growing children. In older patients, and especially those who are delicate, it is wise to prevent an unhealthy imagination being wrongly stimulated by reading too many silly stories or by indulging in innumerable day-dreams, and the weaker physically and mentally these patients are, the more carefully

regulated should their lives be as regards both mental and physical work.

12. CATALEPSY

This is a rare affection. It is a hysterical manifestation, in which the limbs remain for a long time in any position in which they are placed.

Etiology.—It forms a part of hysteria, and may occur in all conditions in which hysteria appears, and it is a phenomenon sometimes present in meningitis and apoplexy. It is the condition induced by hypnotism.

Clinical Features.—The attack is sudden; the patient, usually a female, becomes rigid and may remain standing, sitting, or lying in exactly the same position until the seizure passes off. The limbs are at first so rigid as to be governed with difficulty, but soon they become plastic and remain for some time in any position in which they are placed. It is even possible to mould the features so as to make the patient's face demonstrate grief, surprise, or pleasure, and the attack may last for several hours.

The **Prognosis** is that of hysteria and is generally favourable. The **Treatment** consists in antihysterical remedies. Give tonics and endeavour to improve the nutrition of the patient.

13. TRANCE

This is a condition closely allied to catalepsy, in which for hours, days, or even months, the patient may remain rigid and to all appearance in a profound death-like sleep. The breathing is shallow, the pulse feeble, and nervous people imagine that death can be so closely simulated by trance that living persons may be buried alive. In the most marked instances trance has existed for years and the patient is only awakened for brief periods, once more to lapse into slumber. Such cases are rare and nearly always terminate fatally.

In the more ordinary cases where feeding is difficult, where there is insensibility to pain, and sometimes even temporary suppression of the urine, it is wise to send the patient to a home or asylum, because the attack closely simulates a hysterical manifestation, and the treatment suggested not being appreciated by the patient is generally promptly successful.

VIII. VASOMOTOR AND TROPHIC DISEASES PROBABLY REFERABLE TO THE NERVOUS SYSTEM

1. ANGIO-NEUROTIC (EDEMA)

A transient form of local oedema, often hereditary, and which has in many cases a close relationship to attacks of colic.

Etiology.—It is sometimes associated with urticaria, sometimes with Raynaud's disease, and less frequently with purpura. It is impossible to account for all cases, and the condition may develop without apparently the remotest reason.

Pathological Anatomy.—It seems probable that it is due to a vascular lesion which permits of the transudation of lymph through the vessel walls, but the fact that it is a temporary condition greatly adds to the difficulty of discovering its exact nature.

Clinical Features.—A common site for the oedema is the face and especially the eyelids. The patient may awaken in the morning with both eyes so swollen as to prevent the possibility of the lids being opened, and similar oedema may involve the lips, cheeks, hands of the hands, the throat, and especially the larynx, the legs, and indeed, almost any part of the body. The attacks are apt to come on at some particular time of the day, and they generally last for a few hours. There is often intense itching, and in the throat, in which case the swelling is usually confined to the larynx. The attacks may recur every few weeks or months, and they may be induced by a variety of causes. They sometimes develop during pregnancy, and are present during most of the pregnancy, and they may be present at yet other periods of pregnancy. The attacks may be induced by a variety of causes, and they may be induced by the use of certain drugs, and they may be induced by the use of certain foods, and they may be induced by the use of certain medicines. The attacks may be induced by the use of certain drugs, and they may be induced by the use of certain foods, and they may be induced by the use of certain medicines.

The Prognosis.—The prognosis is usually good, and the attacks are usually self-limiting, and they may be induced by the use of certain drugs, and they may be induced by the use of certain foods, and they may be induced by the use of certain medicines.

The Treatment.—The treatment is usually good, and the attacks are usually self-limiting, and they may be induced by the use of certain drugs, and they may be induced by the use of certain foods, and they may be induced by the use of certain medicines.

and try the effect of administering one of the nitrite group, and particularly nitro-glycerine.

2. RAYNAUD'S DISEASE

(SYMMETRICAL GANGRENE, LOCAL ASPHYXIA)

As the title implies, this disease is a form of symmetrical asphyxia, sometimes resulting in gangrene; it is due to vasomotor spasm followed by vasomotor paresis.

Etiology.—The *predisposing* causes appear to be the existence of some nervous disease, severe anæmia resulting from malaria, scleroderma, and injuries to the solar plexus and splanchnic ganglia, while the *exciting* cause is cold, often associated with excessive fatigue.

The disease is more common in women, it is sometimes directly associated with hysteria, and in a group of cases it is found to be congenital and sometimes hereditary.

Pathological Anatomy.—There is little doubt that a form of peripheral neuritis is present in not a few cases, and with this there have been described definite changes in the arteries and veins, which, however, appear to be more in the direction of spasm, followed by dilatation, than in any organic alteration. No central lesion, common to a number of cases, has as yet been discovered.

Clinical Features.—The fingers, toes, and more rarely the nose and ears are the parts affected. They become during the *first stage* numb, waxy-looking, and bloodless—the condition generally described as *local syncope*. During this stage the fingers feel numb and may tingle, while at the same time the patient complains of chilliness or nausea. The stage lasts for a few minutes or a few hours, and, when it passes off, is apt to be replaced by the *second stage* or the stage of *local asphyxia*, in which the spasm of vessels relaxes, the vessels dilate and the affected parts become livid. This stage is accompanied by more or less pain, sometimes agonising in character, at other times merely a tingling sensation. In a limited number of instances a *third stage* is reached, namely, *local gangrene*, due as a rule to the circulation not becoming re-established, and the tips of the fingers or part of the nose or ears may slough off.

In certain cases either the first or second stage may be

specially marked, while the third stage is rare. Paroxysmal hæmoglobinuria is not infrequently associated with the attacks, and various other phenomena may develop; these include the presence of sugar in the urine, the association of scleroderma or erythema, and sometimes amblyopia and other eye changes. Nervous phenomena have also been described as being occasionally present, such as hemiplegia (sometimes with aphasia), coma, great depression, and more rarely mania. The blood in many cases is considerably altered, the corpuscles failing to run into rouleaux.

The typical attack, with or without paroxysmal hæmoglobinuria, and with one or more of the other symptoms just mentioned, may be induced by exposure to cold, provided the individual is susceptible.

The **Prognosis** depends on the severity of individual cases. The disease may be eventually fatal from spreading gangrene or from intercurrent affections, but it is not infrequently amenable to treatment.

The **Treatment** consists in prophylactic measures to prevent attacks rather than in the use of any special curative drug. The clothing should be warm, the parts affected should be wrapped up in cotton wool, and probably there is no remedial agent so valuable as galvanism applied to the fingers or toes while immersed in water. Nitro-glycerine and the nitrite group should be tried, and for the pain associated with the second stage anodyne remedies are of value, and among the best of these are subacetate of lead lotion, and extract of belladonna in glycerine or vaseline.

3. ERYTHROMELALGIA

(RED NEURALGIA)

This form of neuralgia has been mentioned along with neuralgia proper. There are three typical clinical features—pain, redness, and swelling. It affects the hands and feet and appears to be induced by cold, although this is not constantly the case. In one patient the attack was induced by continuously working in cold water during the winter months: in other cases cold relieves and hot weather makes the disease worse. The pathology of the condition is open to question,

but it seems probable that debility together with a neurotic constitution may be predisposing factors. It is said to be the result of a neuritis, but this seems improbable.

In addition to the three phenomena just mentioned there are not infrequently definite trophic changes induced in the skin and nails of the affected limb, while the temperature of the part may be considerably heightened. Sometimes vesicles appear on the fingers or toes, but these are not common, and there may be considerable alteration in sensation both in the direction of anæsthesia and hyperæsthesia.

Diagnosis.—The condition is not the same as *Raynaud's disease*; there is not the local syncope stage during which the affected part is deprived of blood as the result of vascular spasm, and in quite a number of cases of erythromelalgia the affection is not symmetrical, and there is no hæmoglobinuria, even occasionally, associated with it.

Prognosis.—Different cases vary with regard to duration. The attacks may pass off quickly, or on the other hand they may remain and recur, at intervals, for many years.

Treatment.—Probably rest and supporting the affected limb are the best methods to adopt. Care should be taken to prevent exposure to cold, where cold makes the condition worse. Various local remedies have been used for the relief of pain, such as the hypodermic injections of morphia or cocaine.

4. SCLERODERMA

This disease is certainly one coming under the group of tropho-neuroses. It belongs more properly to the Integumentary System, but inasmuch as, associated with a diffuse thickening of the deeper portions of the skin, there may be also similar changes in the muscles, a brief reference to the disease is desirable. It is more common in females than males. It may be limited, when the term *Circumscribed* has been applied to it, or it may be *Diffuse*, affecting the whole of the skin and greatly interfering with the usual movements.

In the *Circumscribed Type* the affected areas, often of small size, are brawny or hide-bound; they are sometimes pigmented, or in other cases show complete loss of colour. These patches are most frequently found on the neck or chest

and have a peculiar life-history, sometimes appearing and then disappearing in a remarkably rapid way.

In the *Diffuse Type* the whole skin is hide-bound and is firmly adherent to the subjacent tissues. Movements become difficult, the face loses its expression, and various changes in the local circulation may be associated. Raynaud's disease has been described as coincident in several instances of diffuse scleroderma.

The skin is sometimes typically waxy in appearance. In other cases ulcerations or bullæ develop, and there are a considerable number of modifications of the disease which may be met with.

The **Treatment** is unsatisfactory. Probably hot baths, massage, and in certain cases the administration of thyroid extract will afford considerable relief.

5. OSTEOMALACIA

(MOLLITIES OSSEUM)

A rare disease which, in its typical form, occurs in adults, and consists in the softening of certain of the bones due to actual decalcification.

Etiology.—It is a disease of early adult life; it is commoner in women, and in them is associated in some way with pregnancy.

Pathological Anatomy. The central parts of the affected bones are decalcified, leaving only an outer shell which readily yields under pressure, and may be easily cut with a knife.

Clinical Features.—The pelvic bones, the long bones of the legs, and to a less degree those of the arms, are specially affected, and marked distortion of the skeleton and shortening of the patient's stature result. The disease may be slowly progressive, lasting for years, and if the ribs and spinal column are involved, much misery ensues. Lime salts are said to be excreted in the urine during the progressive stages of the disease.

It is incurable, and there is no treatment, unless symptomatic.

A somewhat similar softening of the long bones and ribs may occur in the young, but it is extremely rare.

6. ACROMEGALY

An affection in which portions of the body, especially the hands, feet, lower jaw, and more rarely other parts, become enlarged, sometimes limited to one side of the body.

Etiology.—It occurs in both sexes and in all races, and generally begins about puberty. It is apparently related in an indirect way to syringomyelia, and more closely to hypertrophic osteo-arthropathy and gigantism; while rheumatism, mental worry, syphilis, and the continued fevers are said to have had some connection with recorded cases.

Pathological Anatomy.—The most constant change is found in the pituitary body, which may be hypertrophied, cystic, or the site of a tumour. There is a true hypertrophy of the affected bones, the increase being sub-periosteal.

Clinical Features.—The disease often begins with debility, headache, and sometimes changes in sensation. Sweating is frequently noticed, and occasionally pigmentation of the skin. The typical feature is an enlargement of bones; the affected part begins to enlarge, but is not deformed, and can be freely used. The arms and legs escape in cases in which the feet or hands are enlarged, and in the foot the big toe may be proportionately much larger than the other toes. Where the face is involved, the head does not take a corresponding part in the enlargement; the lower jaw may become enormous, and sometimes the face as a whole is affected. The gait is apt to be slouching and slow, spinal curvature generally develops, and there may be changes in the skin which suggest that the thyroid gland is implicated, and this is not infrequently the case. The mind often begins to fail, the speech becomes slow, and the patient not infrequently shows considerable irritability of temper.

The **Prognosis** is not unfavourable, although life is rarely greatly prolonged, and the treatment is extremely unsatisfactory.

Treatment.—Pituitary extract has been administered, and also thyroid extract, but apparently without much success.

7. HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

This consists in enlargement of the hands and feet and the lower ends of the long bones, but the head does not

suffer. It is associated with long-standing pulmonary disease, sometimes chronic bronchitis, empyema, asthma, or tubercular affections. The disease occurs in adults, and generally in males, and beyond the association of the enlargement of bones with the pulmonary affection the etiology is obscure.

8. OSTEITIS DEFORMANS, OR PAGET'S DISEASE

This is another rare disease, involving the shafts of the long bones, and also sometimes the cranial bones. The bones enlarge and soften, and become curved and misshapen from the weight of the body, frequently causing very great deformity. It seems not improbable that there is a relationship between this affection and malignant tumours of bone.

9. FACIAL HEMIATROPHY

In this peculiar affection there is wasting of one side of the face. The disease commences in childhood as a general rule. It may begin at one spot and gradually spreads, involving the skin, the bones, and to a less extent the muscles. This wasting is sharply limited to one-half of the face, and the upper part appears to be more affected than the lower. The hair and skin often change colour, and the teeth may fall out; sometimes the tongue participates in the hemiatrophy.

The pathology of the disease is very unsatisfactory. It has been considered to be due to an affection of the 5th nerve, and to involve specially the superior maxillary division.

A limited degree of asymmetry of the face is not uncommon, and is often associated with congenital wry-neck, scleroderma, infantile hemiplegia, and some other conditions.

DISEASES OF THE MUSCLES

(1) MYOSITIS

INFLAMMATION of muscular fibres. Many conditions may produce inflammation of muscles, as, for example, the presence of suppurative organisms from whatever cause, the existence of gummata, and possibly tuberculosis. Trichinosis is also a localised myositis, the encysting of the embryo trichina causing a localised inflammation of the muscle. There are, however, cases in which the muscles suffer from a marked and direct fibrosis, the fibrosis being often secondary to, or associated with, a similar involvement of the deeper layers of the skin, and therefore related to scleroderma.

(2) MYOTONIA CONGENITA (THOMSEN'S DISEASE)

A peculiar rigidity of voluntary muscles which comes on when an effort is made to contract the muscles. The condition is transient, and only returns after a period of rest. It was described first by Thomsen, who was himself a sufferer from the disease, and in whose family it had existed for several generations.

Etiology.—The disease is a hereditary one. It shows itself in childhood, and males suffer rather more than females. It is commoner in certain countries, especially Norway, Sweden, and Germany, but it is a rare disease.

Pathological Anatomy.—Portions of muscle which have been harpooned out during life show, according to some authorities, indistinct transverse striation, while the sarcolemma nuclei proliferate and the fibrils hypertrophy.

Clinical Features.—A tonic spasm of voluntary muscles occurs on commencing any muscular effort; it is best marked after rest, but the spasms soon pass off if the muscular effort

is persevered in, and do not recur till after another period of rest. The spasm may begin when the patient endeavours to walk, but after trying for some little time he walks well for hours with no return of the spasm. The legs suffer more than the arms, and very rarely are the face muscles involved, although, apparently, in some cases few muscles can be said to be entirely free from the affection. The spasms increase under certain conditions, such as cold, damp, great heat, excitement, etc. Tapping a muscle causes a groove which lasts for some time, and, as one would expect, there is increased irritability to faradism, while what Erb has termed the myotonic reaction is generally present. This consists in the muscle, when stimulated by faradism or galvanism, contracting and relaxing slowly, a wave-like contraction passing from the cathode towards the anode.

There are no sensory changes, and rarely does the patient suffer more than slight, but constantly-recurring, inconvenience from the disease.

Diagnosis and Prognosis. — The diagnosis is easy. Although cure never occurs, the condition is compatible with a long and useful life, and does not as a rule appear to become worse as time goes on.

The **Treatment** is *nil*. The patient learns how to avoid causing the spasms, and this he generally manages to do in an extremely adroit fashion.

(3) PSEUDO-HYPERTROPHIC PARALYSIS

A form of muscular weakness, associated, however, with apparent enlargement of many of the weakened muscles and with atrophy of others. It has for a long time been considered as a disease essentially of the muscle fibres, and not primarily of the nervous system, although in a few instances changes have been described in the motor cells of the anterior horn governing the affected muscles.

Etiology.—It is often hereditary, and is confined mostly to males, although it is transmitted by the female line. The disease commences soon after infancy, or between the ages of 4 and 6, although in some patients it may not develop till about the age of 15 or 16. In many cases no probable cause can be traced.

Pathological Anatomy.—At death the large muscles have generally become somewhat smaller than normal; the muscular fibres are narrowed, and are separated by fibrous tissue and large numbers of fat cells. During life, if a portion of affected muscle is harpooned out, it is found, on microscopical examination, to contain an excessive amount of fat, although the striation of the muscle fibres may be fairly normal. In some cases there is a new formation of fibrous tissue, and not merely an increase of fat. The naked-eye appearance of the muscle shows an excess of fat.

It is difficult to offer a satisfactory theory to explain the condition. It probably implies an embryonic error, and develops when strain is put upon the muscles.

As already stated, no constant changes have been noted in the nervous system to which the disease can in any way be referred.

Clinical Features.—The child appears to have well-formed muscles, but the stout, muscular-looking legs are surprisingly weak, and at an early period in the disease the child finds great difficulty in climbing up stairs. The muscles affected first are the calf muscles, gluteal muscles, infraspinatus, deltoid, etc., and to begin with, these are usually hypertrophied, but the hypertrophy is accompanied by weakness and not greater strength. Other muscles waste, such as the latissimus dorsi, teres major, and the lower part of the pectoralis major, while the biceps and triceps sometimes hypertrophy and sometimes waste. Other muscles escape altogether, such as the forearm and hand muscles, and the muscles which move the toes, but it is generally noted that the leg muscles as a whole are more affected than those of the arm, and that in the arm the muscles above the elbow are almost alone involved.

If the child be laid flat on the floor, and told to assume the erect posture, he has to "climb up his legs," the typical attitudes during the process being very suggestive of the disease. When standing, great difficulty is experienced in bringing the legs together if they are widely separated. The muscular weakness causes marked alteration in gait and posture, including lordosis, and not infrequently contractions occur, such as talipes equinus, while the joints may be ankylosed, necessitating tenotomy, in order to permit the patient to move the limbs. The knee-jerks are gradually lost

as the disease progresses, the electrical irritability to faradism and galvanism is diminished, but as a rule the organic reflexes do not fail until late in the disease. Sensation is unaffected.

Diagnosis.—The age of the patient, and the typical gait and appearance of the hypertrophied muscles, together with the history of several members of the same family being similarly affected, render the diagnosis easy.

Prognosis.—The disease is incurable, and generally progresses slowly, but surely, until the patient is bed-ridden. Death occurs from some intercurrent affection.

Treatment.—Little can be done by way of treatment, no drug affording any particular benefit. Attention to the general health, massage of the muscles, and tenotomy when necessary, are all the methods of treatment to which we need refer.

(4) IDIOPATHIC MUSCULAR ATROPHY

It has been suggested that pseudo-hypertrophic paralysis, and the juvenile and hereditary forms of what are here called idiopathic muscular atrophy, may be in reality different types of one condition. But to avoid confusion, and with the full recognition that there may really be no true subdivision, a brief description is given here of idiopathic muscular atrophy separate from pseudo-hypertrophic paralysis.

In the idiopathic forms there is no muscular enlargement, and wasting is the chief and typical condition.

In the *Juvenile Type* of Erb the condition closely resembles pseudo-hypertrophic paralysis in distribution, only there is an absence of muscular enlargement; while in the *Facio-Scapulo-Humeral Type* described by Landouzy and Déjerine there is, as the name implies, an involvement of the face muscles along with those of the shoulder. There are many intermediate forms, but these two types predominate.

Etiology.—In both these two types there is a family tendency to the disease, and it may occur not merely in a number of cases in the same family, but also in successive generations. Otherwise there is no recognisable cause. Both sexes suffer, and the age at which the disease commences varies from childhood to late adult life.

Pathological Anatomy.—The appearance of the muscles is similar to that found in pseudo-hypertrophic para-

There is eventually a disappearance of the transverse striation of the muscle fibres.

Clinical Features.—The onset is gradual, and begins with weakness and wasting of the muscles. In the *Juvenile Type* the disease corresponds, as already noted, to pseudo-hypertrophic paralysis, but without hypertrophy of the muscles. In the *Facio-Scapulo-Humeral Type* the face is affected, and the zygomatic muscles are specially involved, causing great alteration in expression, with loss of the naso-labial fold, projection of the lower lip, and an inability on the patient's part to pronounce labials or to whistle. Other face muscles may also be affected.

The electrical irritability of the muscles is diminished, corresponding to the degree of wasting. Occasionally shortening of muscles has occurred, but the organic reflexes and trophic and vasomotor functions are unaffected.

The disease often lasts for many years, and death is invariably due to some intercurrent affection.

Diagnosis.—The family history, together with the distribution of the muscular wasting, generally prevents a mistake in diagnosis, and obviates confusion with *progressive muscular atrophy*.

Prognosis.—The disease is unfavourable as regards cure, but favourable as regards long life.

Treatment.—No drug, so far as we are aware, has any beneficial effect on this disease, but obviously electrical treatment and massage should be given a thorough trial.

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